U.S. NAVY MEDICINE

May-June 1985

U.S. NAVY MEDICINE

Vol. 76, No. 3
May-June 1985

Surgeon General of the Navy
VADM Lewis H. Seaton, MC, USN

Commander
Naval Medical Command
RADM William M. McDermott, Jr., MC, USN

Public Affairs Officer
LT Alan P. Goldstein, USN

Editor
Jan Kenneth Herman

Assistant Editor
Virginia M. Novinski

Editorial Assistant
Nancy R. Keesee

POLICY: *U.S. Navy Medicine* is an official publication of the Navy Medical Department published by the Naval Medical Command. It disseminates to Navy Medical Department personnel official and professional information relative to medicine, dentistry, and the allied health sciences. Opinions expressed are those of the authors and do not necessarily represent the official position of the Department of the Navy, the Naval Medical Command, or any other governmental department or agency. Trade names are used for identification only and do not represent an endorsement by the Department of the Navy or the Naval Medical Command. Although *U.S. Navy Medicine* may cite or extract from directives, official authority for action should be obtained from the cited reference.

DISTRIBUTION: *U.S. Navy Medicine* is distributed to active duty Medical Department personnel via the Standard Navy Distribution List. The following distribution is authorized: one copy for each Medical, Dental, Medical Service, and Nurse Corps officer; one copy for each 10 enlisted Medical Department members. Requests to increase or decrease the number of allotted copies should be forwarded to *U.S. Navy Medicine* via the local command.

U.S. NAVY MEDICINE is published from appropriated funds by authority of Department of the Navy, Naval Medical Command, in accordance with Navy Publications and Printing Regulations P-35. Second class postage paid at Philadelphia, PA, and additional mailing offices. ISSN 0364-6807. Articles, letters, and address changes may be forwarded to the Editor *U.S. Navy Medicine*, Department of the Navy, Naval Medical Command (MEDCOM 00D4), Washington, DC 20372-5120. Telephone (Area Code 202) 653-1237, 653-1297; Autovon 294-1237, 294-1297. Contributions from the field are welcome and will be published as space permits, subject to editing and possible abridgment.

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402.

NAVMED P-5088

POSTMASTER: Send change of address orders to U.S. Naval Publications and Forms Center, ATTN: Code 306, 5801 Tabor Avenue, Philadelphia, PA 19120.

From the Commander
1 We Do Know

Feature
2 Insuring Success to the Most Qualified
CAPT R.E. Cassidy, DC, USN

Hey Doc
6 EMT Test

Interview
10 A Corpsman Remembers Vietnam
D.M. LaMacchia

Clinical Notes
14 Analgesic Therapy for Dental Pain: Salicylates and Para-aminophenols
CDR G.T. Terezhalmy, DC, USNR-R
L.A. Rye, M.S.T., D.D.S.

Professional
19 Synopsis of Diving Medicine for Emergency Physicians (Conclusion)
CDR D.C. Arthur, MC, USN

Scholars' Scuttlebutt
29 Internship Selections . . . Deferments . . . General

Notes and Announcements
9 Self-Study Courses Available for Officers

COVER: HMCM Thomas Eagles dresses a laceration during a 1982 field exercise with the 4th Marine Regiment on Okinawa. Eagles' experience as a corpsman in Vietnam is the subject of an interview on page 10. Photo courtesy HMCM Eagles.

From the Commander

We Do Know

One of the frequently voiced complaints I hear is, "You people in Washington don't really understand what is going on." Implicit in these pejorative words is that those of us in the leadership of Navy medicine have failed to understand the dilemma imposed on those of you "in the trenches." That dilemma is our two-dimensional mandate to support our wartime contingency role and, at the same time, provide an affordable, peacetime health care system. This system itself is supposed to offer almost unlimited access, a constantly expanding diagnostic and therapeutic capability, and be of a quality we can be proud. Moreover, it must satisfy both the self-imposed and external expectations of responsibility and accountability.

The truth is that we do know and understand the problem. As one of my staff recently wrote, "We are confronted with what appear to be blatant irrationalities. We sometimes do not have the resources to do what is expected of us. We are asked to increase our productivity in the face of resources inadequate to do what we were doing previously. We do not have the right mix of people nor do we seem to be able to influence the process that provides the mix we do receive. It is all very frustrating." It is frustrating and we know it. It is almost as if we were told to build a single ship and use it on alternate days as an attack submarine and an aircraft carrier.

It not only is frustrating but also confusing to those of you who must respond to our often inadequately transmitted signals. There is an unavoidable—some might say impossible—conflict between our primary mission of contingency support and wartime readiness and our secondary mission of medical care to an unconstrained constituency. On the one hand we provide constantly increasing man-years of support to our deploying forces, utilizing those skilled personnel whose contingency operational role is well established and with whom, on the other hand, we are trying to increase our in-house ability in order to reduce CHAMPUS expenditures. The efforts of this same group—our surgeons, orthopedists, anesthesiologists, anesthetists, and technicians—is further diluted by their constant involvement in training programs to meet and match our expanding deployable assets.

We know that you have other concerns. Our management process has become increasingly complex. We appreciate the reality that the resources to support these almost revolutionary changes, for the most part, have been drawn from assets formerly devoted to your direct support. We also recognize that their loss has significantly affected your ability to provide care. At the same time we keep saying "do more." Each time we have asked, you have responded. But as someone recently wrote me, "We understand that our priorities were erroneous—not irrational—but inconsistent with the Navy's mission. We really do understand that now. Those who couldn't accept the concept of why the Medical Department exists are nearly gone by attrition. We remain confused that the new requirement appears to be 'we want it both ways,' but with the same or fewer personnel and dollars."

Believe me, we have the message and it is being addressed. From the Assistant Secretary of Defense for Health Affairs through every echelon of the Navy, there is a thorough understanding of the reality of today's environment of competing imperatives and the need to restructure our and your efforts consistent with the resources available. The Assistant Secretary of Defense has directed pilot programs which in part will allow for the purchasing of care from the private sector to augment our capability. At the direction of the Surgeon General, we are exploring the potential for increased contractual support as well as evaluating the consolidation and realignment of some of our services in order to maximize their effectiveness. And always, there is the ongoing, constant, and logical reiteration of our valid needs in support of resource acquisition.

Nothing is easy nor will it be. We respond to direction and mandates we know are too often inadequately transmitted. Navy medical requirements are only a small part of a vast interplay whose ultimate goal is the defense of our country and freedom. As always, your part, your understanding, although seemingly insignificant, will be the critical element in our ultimate success.

William M. McDermott, Jr.
RADM, MC, USN

Insuring Success to the Most Qualified

CAPT Robert E. Cassidy, DC, USN

How many times have you read a selection list and asked, "Why did that board *not* select John Jones? I just don't understand it. He is a superb officer." Worse yet, after reading the same list, you ask, "How did Sam Smith ever get selected? He was only a marginal performer when I knew him and I hear he hasn't improved." If Jones and/or Smith worked for you, indeed you may have contributed to their nonselection and selection respectively. You might not have written a good fitness report on these officers. Not that the evaluation of these officers was not fair, but rather the *quality* of *your* written evaluation was poor.

One of the most peculiar occurrences in life is the failure of the accomplished to succeed or the success of the unqualified. The selection process within the Navy is the best possible system available. The more opportunities that I have to serve on various selection boards, as well as to observe the results of other selection boards, the more confidence I have in the system. Although not perfect, in the vast majority of cases, it works. However, a major weakness in the system is poor quality evaluations written by reporting seniors.

Within the past few years the fitness reports of officers in the Medical Department have gained increased importance. In addition to being reviewed for promotion and augmentation as in the past, they are now reviewed in selecting officers for graduate training, special pay entitlements, retention, assignment, and command. Indeed, the fitness report is the primary tool used for comparing officers and arriving at career decisions with respect to future service. With the recent restructure of the Medical Department including the development of 2XXX billets which are to be filled by the best qualified regardless of corps, it becomes imperative that all reporting seniors have a similar plan in evaluating various subordinate officers.

The purpose of this article is to help improve your ability to write fitness reports. As with any other skill, the writing of fitness reports must adhere to certain principles and rules. Once these have been mastered, this skill is developed to meet an individual's style. However, one becomes proficient only after diligent practice.

Prior Considerations

Unfortunately, many of us have observed in the past the hectic last minute efforts by reporting seniors and administrative staff attempting to complete a fitness report prior to its due date. In contrast, the preparation for writing a fitness report should begin the first day of the reporting period. At a conference with the officer the immediate supervisor should establish performance goals for the upcoming reporting period. It's important to let the officer know what will be expected of him. During the period, the senior should provide continual feedback concerning progress on a periodic basis such as at a monthly conference. Hopefully, more positive than negative comments will be provided at these conferences.

If the officer is not meeting your standards in some area such as grooming regulations, working hours punctuality, or the like, counsel the officer immediately rather than waiting for the end of the evaluation period. If the officer is not counseled at the time these transgressions occur, he/she has every reason to believe that his/her actions are acceptable to you. It certainly is unfair to call an officer to task for unacceptable actions that took place over an extended period when no effort was made to counsel the officer at the time of his substandard performance. In addition, during the evaluation period, present challenges in categories that you will be required to evaluate on future fitness reports. Examples of these challenges are having the officers make oral presentations to in-service training groups that you attend and by having them write a report for your review on a meeting or conference that they attended. Two areas that are poorly judged, particularly with junior officers due to lack of opportunity, are writing and speaking skills. It is important to provide objective criticism and feedback on these oral and written reports.

A confidential personal log should be kept on subordinates. During the evaluation period, enter important items. This provides documentation for the established goals at the start of the evaluation period, verbal compliments received concerning performance, dates and topics of conferences and counseling sessions, your periodic evaluation of performance noting specific accomplishments, and similar entries. In addition, establish a personal file for the officer to include important documents and correspondence such as copies of "atta boys" the command receives concerning the officer's accomplishments, participation in correspondence courses, CME credits earned, specialty board results, etc. Insure that the log and file contain positive as well as negative comments. Remember that the officer has the right to review both the confidential log and the personal file.

Pre-evaluation Review

Before actually sitting down to write the fitness report, the reporting senior should ask himself some very basic questions. Should the officer be in the Navy? Should he be promoted? The answer to these questions are not always simple and lead to

---

Dr. Cassidy is commanding officer of the Naval Dental Clinic, Newport, RI 02841.

other questions. How has the officer's particular department or clinic contributed to the overall accomplishment of the command's mission? How did he contribute? Have all commitments been met in a timely manner? What have been the results of various inspections (IG, JCAH, ADA, etc.) and what role did the officer play? What problems have been encountered and how successfully did he solve those problems while precluding other potential problems? Did he perform significant duties and services that can be recognized in a fitness report or should some further recognition of performance be considered?

The subjective impression of the officer that the reporting senior has developed must now be documented by objective facts. The reporting senior should request input from the officer as in the sample memorandum (Fitness Report Brief Sheet). Hopefully, it is command policy that the officer's immediate supervisor initiates the fitness report. However, if this is not policy, review the evaluation provided by the immediate supervisor and then the input from the officer. Now is the time to review for documentation the contents of the confidential log and the personal file.

Grading

Proper administrative preparation will save much time. Few of us are fitness report experts so request assistance from your administrative staff concerning the latest regulations on fitness reports.(*1-3*) Also review personally timely articles on the writing of these evaluations.(*4-6*)

Take adequate time to prepare for evaluating the officer and for writing the fitness report itself. How much time should this take? As a rule of thumb, the same time you think your reporting senior should take in preparing and writing your own fitness report.

Do not evaluate an officer low on his initial evaluation with the intent of raising his evaluation in subsequent reports. Each report of itself should be able to stand alone. Indeed, it is well within the realm of possibility that an officer deserves to receive a 1 percent fitness report with a recommendation for early promotion on his initial evaluation from you.

In evaluating your subordinates apply uniform standards. Be honest and fair in your judgments. Haering(*4*) discusses very well the dangers of a "false-positive" report. In grading, also be cognizant of the halo effect.

It is imperative to know the climate in which the officer is competing. Official directives(*1-3*) are very explicit in evaluating subordinates so that all these evaluations considered together would fit a bell curve. In real life this does not occur. Unfortunately, inflation and grade creep have affected officer evaluations so that grades are now skewed to the left. You must be aware of what level of evaluation is required for the various types of selection for which the report will be screened. Your ignorance in this area could cause irrevocable damage to an officer's career.

Compare the officer with other officers of:

- the same rank,
- approximately same time in grade, and
- similar competitive category.

Compare:

- Dental Corps officers with Dental Corps officers,
- Nurse Corps officers with Nurse Corps officers,
- Medical Service Corps officers with Medical Service Corps officers, and
- Medical Corps officers with Medical Corps officers.

Appraisal Worksheet

The appraisal worksheet is the guideline for the smooth OCR report on the fitness of an officer. The first portion provides administrative information concerning the officer being evaluated and on the reporting senior. The next section contains in-depth elements for each of the aspects of the individuals performance that you will judge. The marks in these sub-items will provide guidance as to the final grade that should be given in each category and also the strength of that grade (e.g., a "high B" or a "low A") which will be of benefit when you rank the officers judged to receive the same overall grade evaluations. All blocks on the form should receive a letter grade. However, when uncertain of the officer's evaluation in a category due to your lack of familiarity with his performance in a particular area, do *not* assign an arbitrary grade. If an arbitrary grade is assigned, it may be too low which is unfair or may be too high which is unfair to the officer's peers who have legitimately earned that high grade. The only fair evaluation in this instance is to provide a letter grade of "N" (not observed or not applicable). The final section of the appraisal worksheet is the comments section.

Comments Section

The comments section is the most important part of the fitness report. It is in this area that the reporting senior *must* document with supporting evidence the grades previously marked in the various categories. In addition, other information not covered in the graded categories pertaining to performance should be included. Clarity cannot be overemphasized, for this part of the fitness report is subject to the greatest misinterpretation.

In an effort to provide structure as well as uniformity to the fitness reports of officers in the Medical Department, the comments sections should be written in a seven paragraph format.

The first paragraph should set the tone for the rest of the report. The initial statement should be the classical topic sentence providing information as to who, what, when, where, and the officer's overall evaluation.

The second paragraph graphically paints a brief word picture of the individual. What sets this officer apart from his peers?

The third paragraph is an evaluation of the officer as a health care specialist. Comment should be made concerning the quality of the services provided as a clinician, an educator, a researcher, and an administrator.

The fourth paragraph describes performance as a naval officer—both primary and collateral duties. This should not be a "laundry list" of duties, but rather how the officer accomplished each of his assigned duties. Leadership qualities should be discussed in this area.

The fifth paragraph discusses interaction with the civilian community including professional, civic, and educational activities. Indicate professional organizations to which the officer belongs and note any leadership positions held. Also mention professional presentations made to civilian organizations.

The sixth paragraph is a discussion of honors and awards received. Included in this part should be military decorations, letters of appreciation, and civilian honors.

The final paragraph is the most important. Based on the preceding grades and comments, it is here that the reporting senior makes his recommendations concerning the officer's future. Specific recommendations should be made concerning *all* categories for which the officer is eligible, such as special pays, future assignments, retention, administrative assignments, and promotion. If eligible in a category and no recommendation is written, this omission may be interpreted as a negative. It is suggested that the strength of your recommendations in ascending order be:

- not recommended
- recommended
- highly recommended
- very or most highly recommended
- strongly recommended
- very or most strongly recommended

Recommendations

The comments section does not have to be the great American novel, but do make words count and avoid stereotyped phases. In supporting the grades that you made in the previous section, comments should be specific, as well as objective. Selectively underline and use bullets for emphasis. Overuse of these techniques will soon cause them to lose impact. Although you are only required to rank officers evaluated for early promotion, it is highly recommended that in the comments section mention is made of the officer's ranking within a particular grade category. If you do not provide this evaluation, then the selection board will make this determination and their ranking of the officer within a category may not coincide with your actual evaluation.

Insure that specific areas are addressed in the comments section as directed by official references.(*1-3*) For example, these include such topics as the final results of NJP proceedings, UCMJ infractions, an officer's significant weaknesses, and medical conditions necessitating hospitaliza-

Fitness Report Brief Sheet

Date: 1 June 1984

From: Commanding Officer
To: Officers attached to Naval Dental Clinic, Newport

Subj: FITNESS REPORT BRIEF SHEET; COMPLETION OF

Encl: (1) Fitness Report Brief Sheet

1. In order to consider everything which would reflect credit upon your performance, forward via the chain of command to the commanding officer, within two weeks of receipt of this memorandum, *all* items of accomplishment which you think would be appropriate for inclusion in your current fitness report. Enclosure (1) is provided to assist you in organizing this information and additional pages may be utilized.

2. Use extreme care and accuracy in filling out enclosure (1), in order to provide your Department Head, your Clinic Director, the Executive Officer, and the Commanding Officer with as much information as possible in preparing your fitness report. If you are in doubt concerning whether or not to list an item, please include the information in the Fitness Report Brief Sheet. Then your reporting senior can judge the appropriateness of the information for inclusion in your fitness report. Review your curriculum vitae. This is not the time to be humble. Do not underestimate the value of your accomplishments and contributions.

3. The types of information that should be provided in enclosure (1) are listed below. Included are representative examples in each category. *Also* include a brief statement as to the significance or impact of *each* of these accomplishments. For instance, "Fleet Liaison Dental Officer. Since becoming responsible for four ships, the number of the average failure rate of the crews of the four ships has been reduced from 17 percent to 4 percent" or "Selected by Junior Chamber of Commerce as Woman of the Year. I was selected from over 20,000 eligible career business women in the area and was first Naval officer so honored."

a. BRIEF OF ACCOMPLISHMENT
  (1) Branch Director
  (2) Assistant Dental Officer/Endodontics Department
  (3) Chairman, Oral Diagnosis Department
  (4) Finance Officer
b. SPECIAL QUALIFICATIONS
  (1) Diplomate of American Board of Oral Surgery
c. MEMBERSHIP ON BOARDS AND COMMITTEES
  (1) Member of Precious Metal Inventory Board
  (2) Member of Promotion Board to Commander, Dental Corps, U.S. Navy
d. COLLATERAL DUTIES
  (1) Member COM (Open) Advisory Board
  (2) Chairman, Navy Relief Annual Fund Drive
  (3) Fleet Laison Dental Officer

e. SPECIAL PROJECTS COMPLETED OR WORKED ON DURING REPORTING PERIOD

(1) Submitted article, "N-2 is Not for You," for publication in *U.S. Navy Medicine*

tion which may affect the officer's performance. On the other hand, do not discuss topics that should *not* be mentioned in the report such as previous failure for selection for promotion.

(2) Developed a time based "unit system" of appointing patients
(3) Developed curriculum for Red Cross Volunteers
(4) Passed Part I of Exam for Certification by American Board of Endodontics

f. SELF IMPROVEMENT ACCOMPLISHMENTS
(1) Received Master of Arts Degree in Education from Providence College
(2) Attended C-4 course
(3) Certified by American Red Cross in cardiopulmonary resuscitation
(4) Completed Navy Correspondence Course in Operative Dentistry
(5) Attended Continuing Education Course in Oral Pathology at AFIP

g. COMMENDATION AND AWARDS
(1) Awarded Navy Commendation Medal for Service during operation "A Way"
(2) Received Letter of Appreciation from University of Massachusetts
(3) Awarded Honorary Doctor of Science Degree by University of Rhode Island

h. COMMUNITY AND CIVIC CONTRIBUTIONS
(1) Taught CPR in evening at Newport Community Center
(2) Assistant Scoutmaster, Boy Scouts of America—Winter Harbor
(3) Coach, Soccer Team—Duxbury Boys Club
(4) Manager, Little League baseball team—Groton
(5) Director, Brunswick Civic Band

i. ACTIVE MEMBERSHIP IN PROFESSIONAL ORGANIZATIONS
(1) American Dental Association
(2) American Association of Prosthodontists (Vice President)
(3) Gold Foil Study Club—University of Connecticut

j. PRESENTED LECTURES TO THE FOLLOWING GROUPS
(1) New London County Dental Society
(2) Newport County Dental Assistant Association
(3) Annual Meeting of the American Dental Association
(4) Graduate Students, College of Dentistry, Harvard University
(5) South Shore Hospital Staff

k. PRESENTED TABLE CLINICS TO THE FOLLOWING GROUPS
(1) Annual Meeting of New Hampshire State Dental Society
(2) Annual Meeting of Yankee Dental Conference

l. OTHER INFORMATION
(1) Taught Biochemistry at Quincy Community College in evening
(2) Lay Leader at Community Church of Narragansett Bay
(3) Selected by Junior Chamber of Commerce "Woman of the Year"—Newport
(4) President of Ballston Spa High School PTA
(5) Presented Key to City of Portsmouth for outstanding service
(6) Listed in Who's Who in America

m. GOALS FOR UPCOMING YEAR
(1) Attend C-4
(2) Complete Endodontics Correspondence Course
(3) Qualify as CPR instructor

n. FUTURE ASSIGNMENT DESIRES
(1) Department Head on AD/AS
(2) Residency in Periodontics at Bethesda

R.E. CASSIDY

If you are the commanding officer and the majority of the report is based on a director's comments from a remote clinic, then it is appropriate to make a statement to this effect at the conclusion of the comments section.

Although only the most junior officers are required to read and sign their fitness reports, when possible, all officers should be given the opportunity to review each fitness report with their reporting senior. This provides written testimony as to the officer's performance during the past reporting period and also allows the senior to provide guidance for improvement, as well as the setting of goals for the new reporting period.

Another suggestion is a mid-reporting period "command fitness report." The procedure for the "command fitness report" at the midpoint of the reporting period is identical as to that used for the regular fitness report submitted to NMPC at the end of the regular reporting period, except that this report remains at the command. The advantages are that the officer receives a written evaluation of his performance halfway through the reporting period, as well as providing those senior further experience in evaluation procedures and the writing of fitness reports.

Conclusion

Reporting seniors should adequately prepare for evaluating subordinates and follow the recommended guidelines in writing the final fitness report. Only through good quality fitness reports written by conscientious seniors can the Navy, in particular the Medical Department, insure success to the most qualified.

References

1. SECNAVINST 1611.1K
2. OPNAVINST 1611.1M
3. NAVMILPERSCOMINST 1611.1
4. Haering G: Fitness report finesse. *Nav Inst Proc*, Jan 1980, pp 34-39.
5. Sylvester TA: Selection board dynamics. *Nav Inst Proc*, Sept 1983, pp 123-125.
6. Beam HH: Help yourself! *Nav Inst Proc*, Aug 1984, pp 132-134. □

EMT Test

The following test was recently taken by a class of Navy EMT students. See how well you do. Answers on page 17.

Answer questions 1 through 20 using the following key:
A = only answers 1, 2, and 3 are correct
B = only answers 1 and 3 are correct
C = only answers 2 and 4 are correct
D = only answer 4 is correct
E = all answers are correct

Situation for Questions 1 through 9: You are part of an ambulance team called to the scene of an automobile accident which involved a rear-end collision between two cars. At the scene you note that there is one man in the front car, he is lethargic, and there is an odor of alcohol in the car. There are two people in the rear car (neither of whom were wearing seat belts) and they are conscious and complaining of neck pain; the driver is also complaining of severe pain in his right thigh, which is wedged under the dashboard, and says he hit the steering wheel and now has pain in his chest and difficulty breathing. His passenger is a pregnant woman whose head obviously hit the windshield, but she feels fine and doesn't want you to help her but, rather, she is directing your attention to her husband who appears in distress. In addition, one of the onlookers is a 65-year-old man with a history of heart problems and he collapses among the spectators without warning. His wife says that he forgot to take his heart pills today.

1. What initial action would you take if you were in charge of a 6-person EMT crew?

1. Assign specific members of your team to each injured person to perform an initial evaluation of his/her condition.
2. Concentrate on the 65-year-old man who has had an obvious heart attack.
3. Assess the accident scene for potential hazards (e.g., fire) to your crew.
4. Pull the injured victims out of their cars to protect them from further danger.

2. What are the 65-year-old man's *possible* medical problems?

1. Heart attack
2. Fractured hip
3. Vasovagal syncope
4. Stroke

3. What would you do for this man?

1. Ask his wife if he has any other medical problems.
2. Perform an initial assessment for airway, breathing, circulation.
3. Begin CPR immediately if you can't feel a pulse.
4. Give him the medications which he had forgotten to take that day.

4. If this man did not have a pulse, what might the monitor on the Life Pack show?

1. Asystole
2. Premature ventricular contractions
3. Ventricular fibrillation
4. Normal sinus rhythm

5. If you decide to begin CPR, which of the following are appropriate?

1. Establish an airway.
2. Begin CPR with another EMT at a ratio of 15 compressions to 2 breaths.
3. Begin CPR with another EMT at a ratio of 5 compressions to 1 breath.
4. Do a Heimlich maneuver.

6. On assessment of the man in the front car, you find that he has a strong odor of alcohol on his breath, his speech is slurred, he is lethargic but combative, and he has a 4-inch laceration on his forehead. What immediate things would you do for this man?

1. Physically restrain him so that you can perform a thorough examination.
2. Try to calm him by identifying yourself and assuring him that you want to help him.
3. Proceed to the next victim since he is obviously not as badly injured as others.
4. Try to do the most thorough initial and secondary assessment that he will allow.

7. You find that the man in the rear car is conscious, alert, and talking, moving his head from side to side. On

close examination you discover that bright red blood is spurting profusely from his trapped leg, he is having marked difficulty breathing, and you can see his whole chest moving in and out with each breath. What is your first priority for treatment in this man?

1. Insure that he has an open airway and assist his breathing.
2. Make sure that he doesn't have a fractured neck before moving him.
3. Check circulation as part of the initial assessment.
4. Treat the obvious arterial bleeding.

8. While you are treating the man in the rear car, his wife complains of a worsening headache and becomes lethargic. What should you do for her?

1. Ask her to lie down and cover her with a blanket so that she is comfortable.
2. Have another EMT take her out of the car.
3. Ask her to help you take care of her husband to take her mind off the accident.
4. A neurologic examination should be performed for a possible subdural hematoma.

9. If you learn that the woman in the last question is pregnant in her 8th month, what other concerns would you have?

1. What is her due date?
2. Has she had any problems with her pregnancy?
3. Does she have any contractions?
4. Does she have any lower abdominal injuries?

10. Before splinting a fracture, you should:

1. Get the right size splint to reach a joint above and a joint below.
2. Elevate the extremity to help control bleeding.
3. Check for distal neurovascular damage.
4. Apply a tourniquet above the fracture site.

11. In examining the chest of a trauma victim, if you notice unequal breath sounds, a lateral shift in the position of the trachea, and the patient complains of worsening shortness of breath, what should you suspect?

1. A pneumothorax
2. A hemopneumothorax
3. A massive hemothorax
4. A serious chest injury

12. In treating a patient with chronic obstructive pulmonary disease, you should:

1. Not administer 100 percent oxygen.
2. Transport the patient in the most comfortable position which is usually sitting up.
3. Loosen any restrictive clothing.
4. Be calm and reassuring.

13. The diabetic has which of the following problems without treatment?

1. Low blood sugar
2. High blood sugar
3. High level of insulin in the blood
4. Low level of insulin in the blood

14. Which of the following treatments would be appropriate for a patient in insulin shock?

1. Keep the feet elevated.
2. Give a glucose solution by IV.
3. Give a insulin immediately.
4. Give a glucose solution by mouth if he is conscious.

15. Which of the following can cause an "acute abdomen"?

1. Strangulated inguinal hernia
2. Appendicitis
3. Peritonitis
4. Duodenal ulcer

16. In childbirth:

1. Vaginal bleeding of more than 500 cc's requires bimanual examination of the uterus.
2. After delivery the infant should be suctioned, dried, kept warm, and observed for respiratory distress.
3. Immediately after birth, the EMT should put 5-10 pounds of traction on the cord to remove the placenta as quickly as possible.
4. In a normal delivery the mother pushes the infant down the birth canal head first and the EMT guides and supports the infant.

17. Full thickness burns:

1. May be painless.
2. Are also called 3rd degree.
3. Have a charred or waxy appearance.
4. Are the only type of burn with blisters.

18. Signs of internal bleeding are:

1. Weak and rapid pulse.
2. Cold and clammy skin.
3. Decreased blood pressure.
4. Nausea and vomiting.

19. A fall on an outstretched arm can cause:

1. Fracture or dislocation of the elbow.
2. Fracture of the distal radius and ulna.
3. Fracture of the proximal humerus.
4. An indirect injury to the shoulder.

20. An involuntary muscle does which of the following?

1. Causes bronchodilation and bronchoconstriction
2. Controls breathing
3. Causes contraction of the urinary bladder
4. Allows the arm to extend

Match the Following:
(one answer per question, no repeats)

21. Snake bite
22. Petroleum products
23. Alkalies
24. Most medicines

A. Do not induce vomiting
B. Induce vomiting
C. Give lemon juice and vinegar
D. Give antivenin

25. A runner collapses during a race and his skin is red, hot, and dry. What is the most probable diagnosis?

A. Heat exhaustion
B. Heat cramps
C. Heat stroke
D. Heat prostration

26. What is the best treatment for the man in the last question?

A. Keep him warm with blankets until transported to a hospital.
B. Warm soup
C. Cool the body by whatever means available.
D. Give him a high carbohydrate drink.

27. Which of the following is the best treatment for frostbite?

A. Rub the affected body part with snow.
B. Rapidly rewarm by soaking in hot water.
C. Allow the affected area to rewarm on its own.
D. Rub the affected area vigorously with your hands.

28. Which of the following is the best for controlling bleeding in most cases?

A. Elevation
B. Direct pressure
C. Tourniquet
D. Pressure points

29. In a trauma patient, blood or clear fluid coming from the nose or ears indicates:

A. Sinusitis
B. Infection of the mastoids
C. An internal and external ear infection
D. Possible basal skull fracture

30. What is the first thing you should do when setting up an oxygen tank?

A. Open the valve a little to make sure the tank is full.
B. Bang on the side of the tank to make sure it is full.
C. Inspect the valve assembly for dirt, rust, and oil.
D. Remove the tape seal.

31. Which of the following delivers the highest oxygen concentration to a breathing patient?

A. Nasal cannula
B. Partial rebreathing mask
C. Non-rebreathing mask with reservoir bag
D. Duckworth face mask

32. Which of the following is in the correct order for describing seizures?

A. Clonic, tonic, aura, postictal
B. Tonic, clonic, aura, postictal
C. Aura, tonic, clonic, postictal
D. Tonic, aura, postictal, clonic

33. Severe electrical shock can cause what type of arrhythmia?

A. Premature ventricular contractions
B. Ventricular tachycardia
C. Atrial flutter
D. Ventricular fibrillation

Match the Following:

34. Choking
35. Hyperventilation
36. Sucking chest wound
37. Hypoxia

A. Occlusive dressing
B. Heimlich maneuver
C. Oxygen
D. Rebreathing

38. What is the best way to stop bleeding from a large extremity artery so that the patient could be safely transported?

A. Direct pressure
B. Pressure on pulse points
C. Tourniquet
D. Elevation

39. The type of shock from an allergic reaction is called:

A. Psychogenic
B. Hemorrhagic
C. Cardiogenic
D. Anaphylactic

Answer the Following True or False:

40. An unresponsive diabetic should ALWAYS be given glucose initially.

41. The tongue is the most common cause of airway obstruction.

42. Electrical burns are of minor consequence because of their relatively small area of contact.

43. When light enters the right eye, the right pupil normally constricts but the left pupil should not change size.

44. All eye injuries should have a pressure type dressing.

45. Exposed abdominal organs should be covered by dressings which are clean and dry.

46. Second degree burns around the mouth are more important than third degree burns covering the same area of an extremity.

47. A hematoma occurs when small blood vessels are destroyed and the blood seeps into the surrounding tissues.

48. Proper initial care of a patient with a spinal injury includes vital signs.

49. A patient with an extremity fracture will aways have swelling and deformity.

50. Crowning is the time in childbirth when you suck out the baby's nose and mouth. □

Self-Study Courses Available for Officers

Nearly 100 different self-study courses are offered by the Office of Continuing Education at the Naval Postgraduate School in Monterey, CA. These courses, available to officers of all military services, are designed to be completed at the individual's own pace while serving at his/her present duty station. Thousands have made use of this unique opportunity for self-improvement and academic development. Successful completion of selected courses can improve an officer's Academic Profile Code (APC), thereby enhancing the opportunity for selection to a service-sponsored graduate education program. Except for the cost of commercial textbooks, no fees are charged for the courses, and no obligated service commitment is incurred.

Those desiring to transfer academic credit to civilian universities should check with their educational institution before taking NPS courses.

Full details of the study program and enrollment procedures are contained in the current Naval Postgraduate School Catalog of Self-Study Credit Courses. This catalog is available at Educational Services and Navy campus offices throughout the Navy and Marine Corps, or a copy may be obtained by contacting the Office of Continuing Education (Code 500), Naval Postgraduate School, Monterey, CA 93943. Telephone: Autovon 878-2558/Commercial (408) 646-2558.

A Corpsman Remembers Vietnam

Diane M. LaMacchia

HMCM Thomas M. Eagles arrived in Vietnam long before most Americans had heard of it. A Catholic monk at the age of 16, he volunteered to go to Saigon to care for aging French and Vietnamese missionaries who were too old or ill to wash and feed themselves.

That was in 1963 during the early days of the U.S. presence in Vietnam when most of the Americans living in Saigon knew each other. Some of those who met Eagles were moved to seek his advice on their moral dilemmas and personal problems.

The young monk felt inexperienced and unprepared to counsel others. Uncomfortable in his role as spiritual guide, he left the monastery at age 18. Two weeks later, in search of the experience he felt he lacked, he joined the Navy.

As a corpsman, Eagles found an abundance of opportunities to learn about people and empathize with them. He returned to Vietnam in 1966, flying medevac, building dispensaries, and advising Republic of Vietnam special forces. His intense involvement with the well-being of others led him to risk his life for them.

Today, HMCM Eagles is one of the most decorated active duty corpsmen in the Navy. He wears the Distinguished Flying Cross for assisting a wounded medevac helicopter co-pilot fly to safety after the pilot's death. He was awarded the Navy and Marine Corps Medal for going into an active Viet Cong minefield to save a child whose leg had been blown off. He received three Purple Hearts, two Bronze Stars, and numerous other medals, including 14 awarded by the Government of South Vietnam.

HMCM Eagles returned to Vietnam to help in the last days of the U.S. presence there and was among the last Americans to leave Saigon from the roof of the American Embassy in April 1975. In 1979 he was asked to serve as one of Vice President Walter Mondale's interpreters at the 1979 Vietnamese boat people's conference in Geneva, Switzerland.

Eagles met his wife, Karin Chùng Tran, in Saigon, where she worked with him as a nurse in the Vietnamese Public Health Service during the war. They live in Fredericksburg, VA, with their two sons, Kevin, 12, and Patrick, 10. The boys and their father share an unusual hobby—collecting and restoring antique railroad cars and locomotives. Two of these 16-ton "toys," an 1894 caboose and a 1921 industrial switcher, are parked in the backyard of their Fredericksburg home. The family plans to lay railroad track and actually run the train around the yard.

U.S. Navy Medicine *spoke with HMCM Eagles at the Naval Medical Command headquarters, where he is involved in the development and assessment of education and training programs for corpsmen and dental technicians.*

USNM: One positive product of the Vietnam war was the development of rapid air transportation for medical emergencies. Techniques used to

Photos courtesy HMCM Eagles

As medical advisor to Quang Xuyen District, Eagles inspects a dressing change while making ward rounds . . .

Diane M. LaMacchia is deputy public affairs officer at the Naval Medical Command (MEDCOM-00D4-1), Washington, DC 20372-5120.

rescue the wounded have since been adopted by civilian hospitals to save the lives of thousands of Americans every year. When you returned to Vietnam in 1966 as a corpsman, how sophisticated was our system of helicopter evacuation?

HMCM Eagles: When I first went there, we had some very old and slow helicopters, the CH-34's (Sikorsky Sea Horses). How we got in and out was not very refined and we got shot down a lot.

In Korea they had used two-man choppers. We went to a bigger chopper and medevac got more refined during Vietnam. We went from these big, old, slow choppers to the Bell Helicopter Hueys and the CH-46's (Boeing Vertol Sea Knights), which were the main Marine medevac choppers.

The CH-46's were more heavily armored than the CH-34's. They had more pilot and crew protection, were faster and more powerful, and could carry more people. They were easier to load because they had a rear ramp.

Later, when I went into the delta south of Saigon, I worked with the Army, and they used Hueys. The Hueys were lower to the ground so the pilots were lower too. In the CH-34's we flew originally, the pilot was about 13 feet off the ground, easier to see and much easier to hit. When the Huey got on the ground the pilot could hide in the elephant grass and was much less of a target. Also, the helicopter was lower to the ground and easier for us to get in and out of and load patients. On the CH-34 you had to lift the patient up three and a half feet, while on the Huey it was about two feet, which made it easier to get in and out, especially if you were sinking in the mud. And we could get in closer to certain places—like the side of a mountain—putting just one skid down and hanging one out. That developed during the war and we got better at it.

We got so good at it that the corpsman on the ground hardly had time to bandage the wounded before we were pulling them off and out to the hospital ships or the two medical battalions, the Bravo Med and Charlie Med. We overflew battalion aid stations for the most part; we just went in and dumped the wounded right off from the battlefield in 10 or 15 minutes.

. . . and inspects a public health medevac boat with the chief of Quang Xuyen District.

Ten or 15 minutes from the time you picked them up?

Yes, and sometimes that might be 5 to 10 minutes from the time they got wounded, if we were in the area. Sometimes we didn't even have a chance to start the IV's.

What other equipment did you carry?

The corpsman on the ground carried a medical bag called a Unit I. Later on, the Unit I became known to the enemy so the corpsman couldn't carry it. The bag identified him as a corpsman and the enemy would want to shoot him. The Viet Cong's idea was to get the officer, get the radioman, get the corpsman—if they got that, they got the leadership, the communications, and the medical—and that's morale.

So the guys on the ground started carrying medical gear in their pockets and there was a limit to how much they could carry. We carried more gear on the choppers, and still couldn't carry a whole lot, because sometimes we didn't have room.

How many people did you have in the helicopter?

One corpsman.

One corpsman and the guy who's driving?

You have two guys driving—the pilot and the copilot—and a machinegunner, a crew chief who fired the other machinegun, and the corpsman. You could get three of four patients in there. Once we got out of the zone where we might get shot at, the others did what they could to help with the patients.

You had one corpsman on the ground?

It depended on how big the unit was. In Vietnam it got to be about 1 corpsman to 10 or 20 marines. In Recon, reconnaissance operations, it might be one to five—four or five marines out there with one corpsman. In platoonsize or company operations there might be 1 to 20. Sometimes the ratio was better than that and sometimes we lost people and it got worse.

What was the maximum number of wounded you could pick up?

It depended on what the day was like. If the weather was hot we might get off the ground with two or three. If it was cold the chopper could pick up more because of the density of the air, maybe five or six. It also depended on who the pilot was and what the terrain was like. The bigger choppers, like the CH-46 or the Army's CH-47 could hold more.

How did you actually pick up the wounded?

At school they taught us to use stretchers—army litters. We rarely used them in Vietnam. We didn't have time to mess with stretchers, we just flung the guys on. The corpsman would go out with a poncho or a piece of canvas and load them on. I can remember picking a guy up by his flak jacket and just hauling him to the back of the chopper.

These guys didn't mind. They might be terribly wounded—missing a leg even. One time we went into a zone where there were a lot of wounded. We went in with '46's, which are bigger helicopters and can hold about 16 wounded, and as we went in, these guys were crawling to get on the helicopter. When we got out to the *Repose* (hospital ship), they crawled out and said, "Drag me, Doc. Just get me off so you can go back and get my buddies." And we dragged them out by their uniforms.

What kinds of situations were you pulling people out of?

It could be a mine incident. It could be a real battle, where people were actually shooting each other versus mine warfare. It could be accidents, heat stroke, or heat casualties.

Heat casualties were interesting because we could get them up off the hot ground and cool them off. We used to strip the doors off the choppers and you'd rarely find a chopper in Vietnam with any windows left. The first thing the marines would do, even on a troop lift, was to take a rifle butt and knock out the windows so they could shoot from them on our way in and out. After a while nobody put windows in choppers and not many doors either.

Of course, this cooled the patients down. Sometimes in the winter—the relative winter, the rainy season when it goes from 110 degrees to 60 degrees—we'd bring patients on and the lack of doors made it very, very cold. Then we'd have to watch out for shock.

I remember one time corpsmen were told to strip their patients and do as much of a body exam as possible before bringing them in because they might find other wounds. It made good medical sense, but when you got in the air without the doors and windows in the rainy season and stripped them, even in the summer, they could freeze up there.

You didn't have time to do it on the ground?

Sometimes not. You asked me how many corpsmen to a unit—1 to 10, 1 to 20. But we went through times when there were very severe shortages of corpsmen because our pipeline couldn't be filled fast enough and a lot of them were killed. The guy's job was dangerous. Mine was hazardous, I guess, but it was really worse on the ground because the corpsman was the guy who had to run up and take care of the wounded.

When you came in on the helicopter, was that in response to a radio call or were you searching around, flying over the area?

Sometimes we were searching, if we were looking for downed pilots or people who didn't have a radio frequency, but normally it was in response to a radio call. Somebody would call you in to come and get them.

Sometimes we weren't really sure who was where; we couldn't really pinpoint them. So we'd say, "Pop a yellow smoke." And sometimes the enemy would be sitting out there, monitoring our radio nets and all of a sudden we'd see two yellow smokes come up. Then we'd wonder which was the real yellow smoke.

We really had to be careful. Sometimes we got sucked into what we called a "false zone," where someone who spoke English very well was with the enemy and talked us down. They'd say, "We're here, come on down," and when we got there the whole world was waiting for us and the helicopter would look like Swiss cheese if we could even get out of there.

Were you always in the air?

For those first few years. Then when I went to Special Ops (special operations) I was on the ground a lot. I was the only American for a long while who worked with the Vietnamese on patrol.

Were all the corpsmen—on the ground and in the helicopters—all trained the same way?

In those days everyone—depending on what year they came in—had 20 or 16 weeks of Hospital Corps school, which in those years was a lot more field-oriented because they knew these guys were going to be out in the field. And then we had field med school, which was 8 weeks.

In addition to that?

Yes. And that was half field and half medicine. "Field" is how to live in a tent, how to run a radio, how to shoot a gun, how to clean a gun. It was like taking a Navy guy to Army or Marine boot camp. Field medical was slightly different. And then, because I was with Recon, I had some other special forces medical training, even though I was in the Navy.

After medevac you went and worked in the villages?

I did that for another year and a half. I built dispensaries for the villages. I trained young men and women—village health workers—in very rudimentary medicine.

HM3 Eagles prepares to move medevac position from Dong-Ha to Hue (1966).

Photo by HM3 Thomas Kelley

HMCM Eagles

Your training as a corpsman enabled you to do all this?

I went to special forces medical training, too, which taught me how to deliver babies. In those days we had the goat lab, where we actually shot the goat or dog and went in and debrided the wound, learned how to tie off the bleeders, set the bone, put drainage in, start IV's, prescribe antibiotics, narcotics, or whatever's appropriate. There weren't doctors where we were.

I delivered babies and also amputated fingers and toes. These people had no other medical care. I had Laotians and Montagnards, Rhade, Rhade-Bru, Gerai, Ko-ho. These were hill and tribes people, not the average lowland Vietnamese—very primitive—and the Vietnamese hated them.

I couldn't put them in a Vietnamese hospital. The Vietnamese just considered them "moi," which is dirt—that's a very bad word—and the moi would not be taken care of. You'd bust your tail to go in and get them out in a medevac, take them down to a Vietnamese hospital, come back later with some more patients for that same hospital, and these Montagnards were lying there still, dying or dead. So if I didn't take care of them, in a very crude way—no Mayo Clinic and no finesse—they'd die. And sometimes they did die because I didn't know enough or have enough talent, but you gave it your best shot.

Were you alone there?

Yes. I was the only corpsman and sometimes I was the only American in the village.

Was that difficult for you?

It wasn't bad. It was kind of a military Peace Corps. We didn't create little Americas. We lived alone in huts, dressed like them, didn't flash a lot of money around. You really watched what you did; you lived on the edge of these people and they were your fence. I was a young enlisted man—E-3, E-4, E-5—at that time, and I made more money than a Vietnamese colonel. Now if you went in there flashing money or acting like some flaming idiot, why should they protect you?

If you're the man or boy next door, then they had some comradeship with you and they'd protect you. And I was protected. The people would say, "Don't sleep in your house tonight; don't go there."

They kept you from harm?

Yes. You might be walking through the village and an old woman would be there washing clothes and she would just look at you. She didn't say, "Don't go," but you just knew. Don't go down the road that way. □

Analgesic Therapy for Dental Pain: Salicylates and Para-aminophenols

CDR Geza T. Terezhalmy, DC, USNR-R
Leslie A. Rye, M.S.T., D.D.S

The most common complaint causing a person to seek dental treatment is pain. Consequently, the primary obligation and ultimate responsibility of every practitioner is not only to restore health but also to relieve pain. Pain is an unpleasant sensory and emotional experience with actual or potential tissue damage. With some exceptions, patients are surprisingly uniform in their perception of pain, although they do differ greatly in their reaction to it. Cultural, emotional, cognitive, and motivational differences will alter or modulate the intensity of a patient's response to noxious stimuli. A level of discomfort that may not require drug treatment in one instance may necessitate extreme therapy in another. Rational analgesic therapy requires an understanding of the patient's needs and a knowledge of the analgesics available for clinical use.

The ever-increasing advertising and promotion of prescription drugs, paralleled by excessive public faith in the efficacy of such drugs, adds to the challenge of managing dental pain. The intent of this article is to share with clinicians information that will help them to select and prescribe the most appropriate dose—soon enough, often enough, and long enough—and to recognize the clinical ramifications of analgesic therapy.

Salicylates

Aspirin (acetylsalicylic acid) is probably the most extensively prescribed and recommended analgesic, antipyretic, and anti-inflammatory agent. It is the standard for the comparison and evaluation of drugs with similar actions. Salicylates other than aspirin are of little clinical value because they are either too toxic for internal use (salicylic acid) or minimally effective (sodium salicylate). Despite its proven efficacy, aspirin has gained a bad reputation with many clinicians and patients. This reputation seems to be based more on an exaggerated notion of the drug's potential adverse effects than on its pharmacological properties. It would be unfortunate to withhold aspirin because of potential adverse effects when a patient desperately requires the relief provided by this most useful analgesic.

Clinical Pharmacological Effects. Aspirin is effective against the dull, throbbing, mild-to-moderate pain of inflammation, in which locally synthesized prostanoids sensitize nerve endings to endogenous pain-producing substances such as bradykinin. Aspirin minimizes inflammation and reduces elevated body temperature without affecting the normal circadian body temperature. It increases bleeding time by preventing irreversible platelet aggregation by interfering with the release of adenosine diphosphate from platelets after primary platelet plugs have been formed.(*1*) Many patients take subtherapeutic levels of aspirin for cardiovascular prophylaxis. These levels may contribute to postsurgical complications, at least in those patients with other bleeding problems. It may be wise to discontinue elective aspirin use 1 week before anticipated surgery. Even in therapeutic doses, aspirin stimulates respiration by increasing the consumption of oxygen and the production of carbon dioxide.

Pharmacokinetics and Metabolism. Aspirin is rapidly absorbed from the stomach and the upper small intestine.

Dr. Terezhalmy is associate professor of the Department of Dental Diagnostic Science at the Naval Dental School, Bethesda, MD 20814-5000, and associate professor of the Department of Pharmacology at the Graduate School of Biomedical Sciences, University of Texas Health Science Center, San Antonio, TX. He was formerly chairman of the Oral Diagnosis Department at the Naval Dental School. Dr. Rye is clinical assistant professor of the Oral Diagnosis Department at Georgetown University, Washington, DC 20057.

It reaches appreciable plasma concentrations in 30 minutes and a peak value at about 2 hours. After absorption, aspirin is distributed throughout most body tissues and fluids, and it readily crosses the placental barrier. Aspirin has a dose-dependent half-life that varies from 2 to 8 hours.(*2*) Its metabolism normally follows first-order kinetics in the liver; however, after large doses, the enzymes that metabolize the drug become saturated, and the half-life increases significantly. Aspirin is excreted primarily by the kidney as water-soluble conjugates.

Dose. Preparations include plain and buffered tablets, buffered solutions, timed-release and enteric-coated tablets, as well as many combination products that include aspirin. The traditional adult single dose of aspirin is 650 mg (two tablets, 10 grains), but the analgesic effect is increased with single doses up to 1,300 mg (four tablets). Single doses larger than 1,300 mg do not increase pain relief and may prove toxic. Differences in the amount of a single dose and the interval between doses have only a minor effect on the steady state of aspirin, which is produced by a daily dosage of aspirin not to exceed 3,900 mg in 24 hours. Therefore, there is reasonable latitude in timing and dose fractionation during a 24-hour period, so long as single doses are taken at least 2 hours apart. The size of the daily dose is critical for maximum analgesia, and it should be individualized on the basis of patient history, age, body weight, and other factors such as liver and kidney function. For children 6 years old and over, the appropriate dosage is 10-20 mg/kg of body weight given in 4-6 doses, with the daily dosage not exceeding 3,600 mg in 24 hours.

Adverse Effects and Contraindications. Intolerance or hypersensitivity to aspirin is most likely to occur in patients with a history of asthma, nasal polyps, or chronic urticaria.(*3-10*) It produces gastric bleeding in patients who use it frequently, causing blood loss in the stool and occasionally an iron-deficiency anemia.(*11-15*) Extensive intrapartum and postpartum maternal bleeding have been noted when the agent is taken within 5 days of delivery.(*16,17*) It is important to note that aspirin is a common component of many over-the-counter and prescription compounds. Because aspirin is a common household drug, it is a common cause of poisoning in young children. A differential diagnosis of adverse reactions to aspirin is presented in Table I.

Para-aminophenols

The two important drugs in the class of analgesics known as the para-aminophenols are acetophenetidin (phenacetin) and N-acetyl-para-aminophenol (acetaminophen). In the past several years both of these agents have received much adverse and favorable publicity. Phenacetin has been indicted as a cause of renal papillary

TABLE I. Differential Diagnosis of Adverse Reactions to Aspirin (*10,18*)

Allergic Intolerance	Mild Salicylism	Severe Salicylism
↑Incidence with	↑Incidence in	↑Incidence in Elderly
Asthma	Young children (accidental)	(accidental
Nasal polyps	Young adults (suicide)	intoxication)
Chronic urticaria		
	Signs/Symptoms	Signs/Symptoms
History (within 3 hours	Tinnitus	Restlessness
after aspirin ingestion)	Dizziness	Garrulousness
Generalized urticaria	Headache	Incoherent speech
Angioedema	Mental confusion, drowsiness	Apprehension
Bronchospasm	Sweating, thirst	Vertigo, tremor
Severe rhinorrhea	Hyperventilation	Diplopia
	Nausea, vomiting	Delirium, hallucinations
Rare hypersensitivity	Diarrhea	Convulsions
Vasomotor collapse	Dehydration	Coma
	Hyperthermia	Death

TABLE II. Acetaminophen Versus Aspirin

	Acetaminophen	Aspirin
Analgesia	+	+
Anti-inflammatory	–	+
Antipyresis	+	+
Antithrombotic	–	+
Onset of action	30 minutes	30 minutes
Half-life	2-4 hours	2.5-8.5 hours
Equigesic dose	650 mg	650 mg
Maximum analgesic dose	Not established	1,300 mg
Maximum daily dose	3,000 mg (adults) 1,200 mg (children)	3,900 (adults) 3,600 (children)

necrosis, interstitial nephritis, renal cell carcinoma, and hemolytic anemia. Lethal doses are characterized by cyanosis, respiratory depression, and cardiac arrest.(*19*) Because most of the ingested phenacetin is metabolized to acetaminophen,(*20*) its active analgesic-antipyretic metabolite, phenacetin should be considered not only a hazard to daily users but also an obsolete drug. The FDA requested the withdrawal of phenacetin from the market as of August 1983. Although the general substitution of acetaminophen for aspirin is not recommended, it is a suitable substitute in patients in whom aspirin is contraindicated.

Clinical Pharmacological Effects. Acetaminophen has analgesic and antipyretic effects that do not differ significantly from those of aspirin.(*21*) It is only a weak inhibitor of prostanoid biosynthesis; it appears to be more effective against enzymes in the central nervous system than against those in the periphery.(*22*) This may partially account for its ability to reduce fever and to induce analgesia. However, its anti-inflammatory effect is weak.

Pharmacokinetics and Metabolism. Acetaminophen is rapidly and almost completely absorbed from the gastrointestinal tract, reaching peak plasma concentrations in 60 minutes. The pain threshold is elevated in 30 minutes with the use of acetaminophen, much the same as with aspirin. Elimination half-life is 2-4 hours.(*23*) The drug is distributed relatively uniformly throughout most body fluids, and evidence of placental transfer has been reported.(*24*) Acetaminophen is conjugated in the liver with glucuronic acid and sulfuric acid; small amounts of other metabolites have also been detected. Elimination half-life is prolonged with liver disease. When high doses are ingested, acetaminophen undergoes N-hydroxylation followed by spontaneous dehydration to form N-acetyl-para-benzoquinone, the metabolite generally believed to be responsible for the hepatotoxicity. After therapeutic doses, 90-100 percent of the metabolized acetaminophen may be recovered in the urine within the first day.

Dosage. Preparations include tablets, an elixer or syrup, capsules, and over 250 combination products containing acetaminophen. The traditional adult dosage of acetaminophen is 650 mg every 4 hours. A ceiling dose yielding maximum analgesia has not been determined, but the drug should not be administered in any amount for more than 10 days. The daily dose should not exceed 3 gm for adults, 1.2 gm for children.(*21,25*)

Adverse Effects and Contraindications. In the recommended therapeutic dosages, acetaminophen is usually well tolerated. An erythematous or urticarial skin rash may occur occasionally, accompanied at times by fever and mucosal lesions. The most serious adverse effect of an acute overdose of acetaminophen, hepatotoxicity, can occur after the ingestion of a single dose of 10-15 gm, and symptoms of hepatotoxicity during the first 2 days of acute poisoning do not reflect the potential seriousness of the intoxication.(*26*) Some special considerations concerning hepatotoxicity include the following:

- Concurrent ingestion of alcohol or other drugs with acetaminophen may enhance its toxicity by reducing the metabolic capacity of the liver.
- Nausea, vomiting, anorexia, diarrhea, and abdominal pain occur during the first 24 hours.
- Clinical evidence of hepatic damage is manifested 2-6 days after ingestion of toxic doses.
- If half-life exceeds 4 hours, hepatic necrosis should be anticipated.
- If half-life exceeds 12 hours, hepatic coma is likely.
- Vigorous supportive therapy is essential when intoxication is severe. It consists of induction of vomiting, gastric lavage, administration of activated charcoal, hemodialysis, and the administration of sulfhydryl compounds, cysteamine, or acetylcysteine (Mucomyst®).
- Recovery after early diagnosis may take 3 months.

Administration of acetaminophen to patients with impaired renal function results in increased accumulation of conjugated acetaminophen in plasma but only minor changes in the plasma concentration of free acetaminophen. A few patients may develop renal papillary necrosis or acute cardiovascular damage.

Conclusion

A comparison of the properties of acetaminophen and aspirin is provided in Table II.

Aspirin is the most extensively prescribed analgesic, antipyretic, and anti-inflammatory agent. It is effective against the dull, throbbing, mild-to-moderate pain of inflammation. The best single dose of aspirin is that which is adequate to relieve pain; the proper dosage interval is that which sustains relief without causing toxicity. It would be unfortunate to withhold aspirin from a patient who desperately requires the relief provided by this most useful analgesic because there have been some adverse effects when it is used improperly. Acetaminophen is a suitable substitute for aspirin for its analgesic or antipyretic uses in patients in whom aspirin is contraindicated (e.g., patients with asthma, peptic ulcer, gouty arthritis, hyperuricemia, and hemophilia or other bleeding disorders, and those taking anticoagulants).*(27)* However, the general substitution of acetaminophen for aspirin as an analgesic in dental practice is not recommended, especially when anti-inflammatory effects are also desired.

References

1. Burch JW, Stanford N, Majerus PW: Inhibition of platelet prostaglandin synthetase by oral aspirin. *J Clin Invest* 61:314-319, 1978.

2. Levy G, Giacomini KM: Rational aspirin dosage regimens. *Clin Pharmacol Ther* 23:247-252, 1978.

3. Samter M, Beers R: Intolerance to aspirin: Clinical studies and consideration of its pathogenesis. *Ann Intern Med* 68:975-983, 1968.

4. Moore-Robinson M, Warin RP: Effect of salicylates in urticaria. *Br Med J* 4:262-264, 1967.

5. Champoin RH, Roberts SOB, Carpenter RG, Roger JH: Urticaria and angioedema: A review of 554 patients. *Br J Dermatol* 81:588-597, 1969.

6. Michaelsson G: Chronic urticaria: A clinical study with special reference to vascular reactions mediated by the mallikrein-kinin system. *Acta Derm Venereol* 49:404, 1969.

7. Settipane GA, Chaffee FH, Klein DE: Aspirin intolerance II: A prospective study in an atopic and normal population. *J Allergy Clin Immunol* 53:200-204, 1974.

8. Settipane GA: Aspirin intolerance: Recent developments. *Cutis* 15:861-865, 1975.

9. Lewis DP, Casterline CL, Stein M: Aspirin idiosyncrasy. *J Am Dent Assoc* 98:737-738, 1979.

10. Abrishami MA, Thomas J: Aspirin intolerance: A review. *Ann Allergy* 39:28-37, 1977.

11. Lanza FL, Royer GL Jr, Nelson RS: Endoscopic evaluation of the effects of aspirin, buffered aspirin, and enteric-coated aspirin on gastric and duodenal mucosa. *N Engl J Med* 303:136-138, 1980.

12. Silvoso GR, et al: Incidence of gastric lesions in patients with rheumatic disease on chronic aspirin therapy. *Ann Intern Med* 91:517-520, 1979.

13. Leonards JR, Levy G: Reduction or prevention of aspirin-induced occult gastrointestinal blood loss in man. *Clin Pharmacol Ther* 10:571-575, 1969.

14. Levy M: Aspirin use in patients with major upper gastrointestinal bleeding and peptic-ulcer disease. *N Engl J Med* 290:1158-1162, 1974.

15. Jick H, Porter J: Drug-induced gastrointestinal bleeding. *Lancet* 2:87-89, 1978.

16. Stuart MJ, Gross SJ, Elrad H, Graeber JE: Effects of acetylsalicylic-acid ingestion on maternal and neonatal hemostasis. *N Engl J Med* 307:909-912, 1982.

17. Rumack CM, et al: Neonatal intracranial hemorrhage and maternal use of aspirin. *Obstet Gynecol* 58:52S-56S, 1981.

18. Anderson RJ, et al: Unrecognized adult salicylate intoxication. *Ann Intern Med* 85:745-748, 1976.

19. *Federal Register*, Tues, Aug 10, 1982, pp 34641-34642.

20. Conney AH, et al: Enzyme induction and inhibition studies on the pharmacological actions of acetophenetidin. *J Pharmacol Exp Ther* 151:133-138, 1966.

21. Koch-Wester J: Acetaminophen. *N Engl J Med* 295:1297-1300, 1976.

22. Flower RJ, Vane JR: Inhibition of prostaglandin synthetase in brain explains the antipyretic action of paracetamol (4-acetamidophenol). *Nature* 240:410-411, 1972.

23. Black M: Acetaminophen hepatotoxicity. *Gastroenterology* 78:382-392, 1980.

24. Levy G, Garrettson LK, Sode DM: Evidence of placental transfer of acetaminophen (letter). *Pediatrics* 55:895, 1975.

25. Hopkinson JH, et al: Acetaminophen (500 mg) versus acetaminophen (325 mg) for the relief of pain in episiotomy patients. *Curr Ther Res* 16:194-200, 1974.

26. Prescott LF, Wright N, Roscoe P: Plasma-paracetamol half-life and hepatic necrosis in patients with paracetamol overdosage. *Lancet* 1:519-522, 1971.

27. Sutor AH, Boure FJW, Owen CA: Effect of aspirin, sodium salicylate, and acetaminophen on bleeding. *Mayo Clin Proc* 46:178-181, 1971. □

Answers from page 6

1.	B	11.	E	21.	D	31.	C	41.	T
2.	E	12.	E	22.	A	32.	C	42.	F
3.	A	13.	C	23.	C	33.	D	43.	F
4.	B	14.	C	24.	B	34.	B	44.	F
5.	B	15.	E	25.	C	35.	D	45.	F
6.	C	16.	C	26.	C	36.	A	46.	T
7.	D	17.	A	27.	C	37.	C	47.	T
8.	C	18.	A	28.	B	38.	C	48.	T
9.	E	19.	E	29.	D	39.	D	49.	F
10.	B	20.	B	30.	C	40.	T	50.	F

Synopsis of Diving Medicine for Emergency Physicians

CDR Donald C. Arthur, MC, USN

Conclusion

In contrast to the relatively straightforward physical nature of barotrauma pathophysiology discussed in part 1 of this 2-part series, the effects of dissolved gases are a bit more complex. The nitrogen and oxygen in the compressed air breathed by SCUBA divers manifest effects because of their increased tissue partial pressures as well as direct effects of bubble evolution caused by liberation of nitrogen from tissues which have become supersaturated during a dive. This article will discuss the pathophysiology, presentation, and initial management of decompression sickness, nitrogen narcosis, and shallow-water blackout. The rationale for substitution of alternate inert gases for nitrogen in the breathing mixture will also be discussed.

Decompression Sickness

Decompression sickness (also known as Caisson's disease, dysbarism, and the "bends") is the result of a series of pathophysiologic responses to the evolution of dissolved tissue gases, precipitated by changes in ambient pressure. Bubbles which are released from solution by a too rapid reduction in ambient pressure either obstruct blood flow, cause blood chemistry changes, or stretch and damage tissue. The symptoms can range from innocuous skin itching to serious central nervous system or pulmonary compromise. Prompt diagnosis and recompression treatment are essential.

On-Gassing and Off-Gassing. Following Henry's law, the rapidity and amount of gas dissolved (or released from solution) in tissues is proportional to the change in ambient pressure. When a mixture of gases is inspired, the amount of each gas which becomes dissolved in tissues is proportional to the partial pressure of each gas. Further, the eventual tissue concentration of inspired gases also depends on the rate at which the gases are either removed or metabolized within the tissues. When breathing air, the dissolved tissue gas of concern is nitrogen since the oxygen component, unlike nitrogen, is rapidly consumed by tissue metabolism. Thus, the major factors which govern dissolved gas accumulation due to changes in ambient pressure are:

- *Ambient Pressure:* Governs the rate and total amount of gas dissolved in tissues. The two most important factors which determine the amount of gas dissolved in the tissues are the depth of the dive and the amount of time spent at depth.
- *Partial Pressure:* Governs the amount of each gas (in a mixture) which is dissolved in tissues.
- *Tissue Metabolism:* Removes oxygen; inert gases and carbon dioxide remain.

In addition, there are many minor factors governing tissue gas dissolution under pressure. Exercise increases gas uptake and elimination by increasing the respiratory rate, cardiac output, and tissue perfusion, thereby increasing the rate of tissue gas delivery and removal.*(1,2,3,4)* Gas and body temperatures and the diver's state of hydration also proportionally affect the rates of gas uptake and elimination.*(5,6)* Smoking and alcohol consumption may increase susceptibility to decompression sickness by a variety of poorly defined mechanisms. Age and sex have also been mentioned as factors influencing the incidence of decompression sickness, but their association probably has limited practical value in sport diving.*(7,8,9,10)*

Under increased ambient pressure, gases become dissolved in different tissues at varying rates since each tissue has its characteristic rate of gas uptake technically known as the "tissue half time." For example, gases become rapidly dissolved in muscle but are dissolved very slowly in bone and fat. Fat can take on a large amount of nitrogen because nitrogen is five times more soluble in fat than in water.*(11,12)* Since gas elimination follows a similar time course, nitrogen is eliminated from fat tissues very slowly, and supersaturation (with subsequent bubble formation) may occur if the ambient pressure is reduced rapidly. Thus, obese people seem to experience decompression sickness

---

Dr. Arthur is senior medical officer aboard USS *Kitty Hawk* (CV-63).

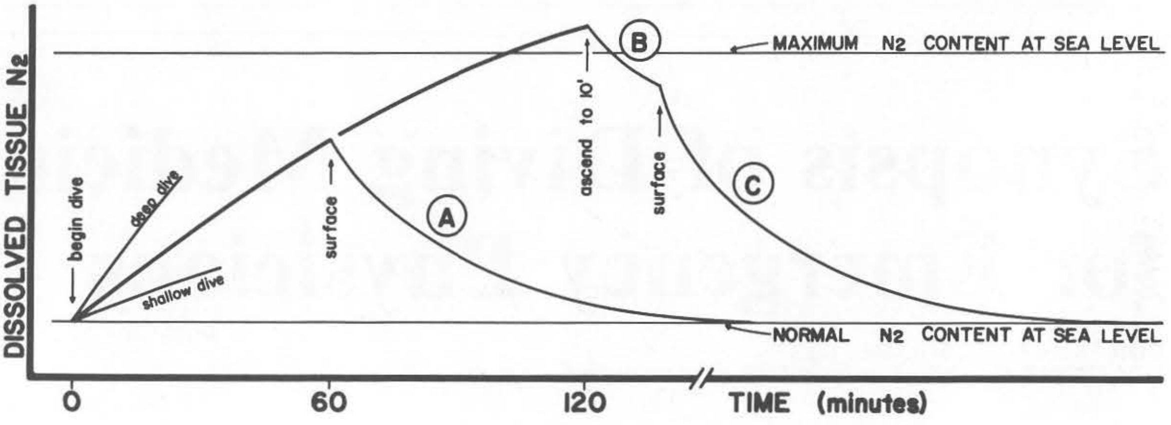

Figure 1. Illustrates the effect of depth on dissolved tissue nitrogen levels. A diver can safely surface directly if the tissue level of nitrogen at depth has not exceeded the maximum level which the tissues can hold at sea level (curve (A)). If this maximum level is exceeded, the diver must ascend to a depth at which off-gassing can occur without bubbling (10' in this case) and remain there until the tissue level falls below the surface supersaturation level (curve (B)); he can then safely ascend to the surface (curve (C)).

more frequently than lean divers.(*7,8, 13,14,15*) This is usually only a factor in longer and deeper dives than those attempted by sport divers, however.

At sea level, gases (mainly nitrogen) are dissolved in tissues at a steady state *minimum* level determined by the three major factors discussed previously. There is also a *maximum* amount of gas which can be dissolved in tissues without evolution of the gas as bubbles. Figure 1 illustrates the gradual uptake of nitrogen during a dive. Gas uptake, or "on-gassing," occurs at a faster rate during a deep dive than on a shallow dive and continues from the start of the dive until the ambient pressure is reduced by ascent. If ascent is delayed, tissue gas levels will continue to increase until the tissue concentration reaches the steady state level consistent with the new, higher ambient pressure. This takes approximately 12 hours at the higher ambient pressure and is only of concern when performing saturation dives wherein divers intentionally stay underwater for many hours or days.

If the diver ascends before the level of dissolved nitrogen reaches the maximum permissible at sea level (supersaturation), then gas elimination, or "off-gassing," will occur gradually and without bubble formation (curve (A)). If the diver were to remain at the increased ambient pressure until the amount of dissolved tissue nitrogen exceeded the supersaturation level, then nitrogen would probably be liberated as bubbles if the diver surfaced directly. The increased ambient pressure at depth keeps the additional tissue nitrogen dissolved, whereas the lower ambient pressure at the surface would promote gas evolution at a rapid rate. In this case, the diver should ascend to a safe depth for a "decompression stop" where the ambient pressure is sufficient enough to allow gradual off-gassing while preventing bubble evolution, yet is not great enough to cause additional gas to become dissolved in the tissues (curve (B)). After the time at the decompression stop has been sufficient to bring the tissue nitrogen concentration below the supersaturation level and the danger of bubbling is minimal, the diver can ascend directly to the surface (curve (C)). Some dives require more than one decompression stop where the first stop is made to allow enough off-gassing for the diver to safely ascend to the next stop.

Although actively discouraged in sport diving because it is extremely complicated and fraught with danger, many casual divers practice decompression diving without appreciating the risks of miscalculation. Sport divers should always follow nondecompression diving tables and ascend before the surface supersaturation level is reached. The U.S. Navy diving tables(*16*) are well recognized and are used as a guide to safe diving time/depth constraints.

Since the amount of dissolved tissue gas gradually returns to the normal sea level amount over the 12 hours following a dive, the times for repetitive dives on the same day must be adjusted. Figure 2 illustrates a nondecompression dive lasting 60 minutes and a second dive which is begun before the tissue nitrogen level has returned to normal. This additional tissue nitrogen remaining at the beginning of a subsequent dive is called the *residual nitrogen.* Since the second dive starts with some residual nitrogen, the time allowed for the second dive should be shortened by the time which would

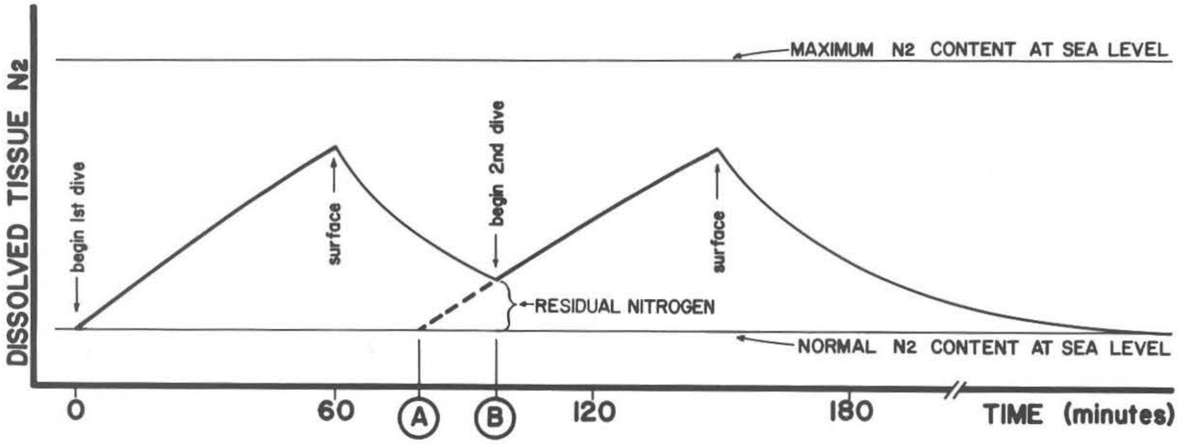

Figure 2. Illustrates nitrogen on-gassing during repetitive dives. If a second dive is begun before the tissue nitrogen accumulated on the first dive has totally off-gassed (approximately 12 hours), then the allowable time for a second dive must be shorted by the amount of time which would have been required to have accumulated the amount of "residual nitrogen" in only one dive. This time is known as the "Residual Nitrogen Time" (RNT) and is the time between points (A) and (B).

have been required (if it were a single dive) to obtain the tissue nitrogen equivalent to the amount left over from the first dive. This is called the *residual nitrogen time* and is the time between points (A) and (B).

Reputable diving schools exhaustively teach the proper use of these tables and calculations for repetitive dives. Divers must always use these tables and follow their calculations. A common diving expression is "plan your dive and dive your plan!" Deviations from established depth/time constraints can easily result in the diver exceeding a tissue supersaturation level and increase the risk of decompression sickness.

Clinical Manifestation. Both the time course and the symptomatology of decompression cover a wide spectrum. Symptoms are evident within an hour of surfacing in approximately 80 percent of patients and within 4 hours in over 95 percent;(*17*) symptoms of decompression sickness arising more than 12 hours after a dive are very unusual. Recall from part 1 that the occurrence of a gas embolism is presumed when the symptoms arise within 10 minutes after surfacing. The most frequent error in treatment of decompression sickness is failure or delay in making the diagnosis. This often leads to delayed treatment, resulting in permanent sequelae instead of prompt resolution.

Decompression sickness has customarily been divided into two categories (Table 1): Type I which manifests as limb pain or with skin or lymphatic involvement, and Type II, also known as "serious" decompression sickness, which encompasses all other symptoms. Although this classification is convenient, decompression sickness is dynamic and a patient initially presenting with Type I symptoms may progress to more serious manifestations. Since treatment of each type is different, careful classification is important.

Type I decompression sickness is the more common variety and describes the classic presentation of "pain only" bends. This includes only pain confined to the arms or legs which may be aggravated by movement and relieved by direct pressure on the area, i.e., with a sphygmomanometer cuff.(*2, 17, 18, 19*) For classification as Type I, there should be *no* association with systemic symptoms and the pain should not be referred or confused with paresthesias or hypesthesias. The pain is usually peri-articular, involves the lower extremities three times more frequently than the upper extremities in divers,(*17, 19*) and ranges from mild discomfort (often called the "niggles") to severe pain.

Skin symptoms are further divided into two groups: pruritis alone and skin marbling (cutis marmorata).(*18, 19, 20*) Pruritis is usually encountered only after deep chamber dives where the diver is surrounded by compressed air and itching usually begins during decompression or shortly after reaching the surface.(*20*) Gas entering the sweat and sebaceous glands during compression is thought to form pruritic bubbles as the diver surfaces.(*20*) The itching of skin bends must be differentiated from the tingling of paresthesias or hypesthesias which are serious symptoms. The pruritis may be somewhat relieved by direct pressure and, although usually localized, may involve a large area such as an extremity or the torso but does not follow dermatomes. Pruritis is not considered a true form of decompression sickness and resolves rapidly without treatment.

Marbling of the skin occurs when subcutaneous bubbles cause venous stasis. The symptoms most commonly occur over the torso and shoulders and commonly begin as intense itching which is followed by discoloration. (*20*) Erythema progresses to cyanotic mottling which may be linear or patchy, is nontender, characteristically blanches on direct pressure, and resolves rapidly when treated with recompression. Marbling *is* a true form of decompression sickness, indicating that tissue supersaturation has occurred, and may be a harbinger of systemic involvement and impending serious symptoms.

Although uncommon, isolated lymphatic symptoms occasionally occur as a result of lymphatic obstruction by bubbles. There is usually pain and swelling of lymph nodes or groups of lymph nodes with variable edema of the tissues normally drained by the affected nodes. Although recompression usually produces rapid relief of the pain, swelling may persist for days.

Type II decompression sickness includes all other manifestations of evolved gas pathology. These are (1) pain elsewhere other than the extremities, (2) any central nervous system sign or symptom, and (3) pulmonary manifestations (chokes). Pain elsewhere other than in the extremities, although not a serious symptom per se, may represent referred pain from visceral sites or spinal cord involvement. Therefore, pain in the head, neck, and torso should be classified as a Type II symptom and treated accordingly while observing for other serious symptoms.

Unfortunately, there is no clear central nervous system symptom complex which is characteristic for decompression sickness. *Any* neurologic symptom can be associated with the intra- and extra-vascular evolution of bubbles anywhere in the nervous system. Spinal cord symptoms are most common in divers while cerebral symptoms predominate in aviation personnel. Paraplegia is the most common presentation of a spinal cord "hit" and may be preceded by ascending numbness or paresthesias or a dermatomal distribution of pain at approximately the level where bubbles have accumulated. Cord lesions are most common in the lower thoracic region and are usually associated with bladder paralysis, urinary retention, fecal incontinence, and, occasionally, priapism.

Cerebral decompression sickness can present with hard signs such as seizures, hemiplegia, or the common visual symptoms of scotomata, diplopia, tunnel vision, or blurring. Headaches are also common and may mimic a migraine headache in severity. Any hard neurologic sign can result from evolution or accumulation of bubbles in any area or near any cranial nerve. Unconsciousness and shock represent global and fulminant manifestations and are distinctly uncommon. Soft signs and symptoms, however, are very common and include unusual fatigue, a sense of detachment from the surroundings (being within one's self or viewing the activities from a point removed from one's body), inappropriate or uncharacteristic behavior, or any number of other variable presentations.

The cranial nerves are not usually affected. If they are, labyrinthine or inner ear decompression sickness (the "staggers") is a common presentation. Either the cochlea or the vestibule can be involved and the patient often presents with any combination of vertigo, nausea, vomiting, deafness, tinnitus, and nystagmus. Immediate treatment of the staggers is extremely important since prolonged obstruction of the small nutrient arteries supplying the inner ear structures could result in permanent damage.

Pulmonary decompression sickness (the "chokes") usually occurs within minutes (rather than hours) of surfacing and presents as a triad of substernal pain, cough, and dyspnea. Substernal pain, usually the first symptom, is described as "burning" in nature, and is aggravated by deep inspiration. The cough is initially occasional but is easily evoked by cigarettes and may progress to uncontrollable paroxysms. The substernal pain increases as the frequency of cough increases and dyspnea becomes superimposed. Although the chokes is relatively uncommon, its severity and prognosis make immediate treatment mandatory. Respiratory failure and shock are often the terminal events in pulmonary decompression sickness.

Pathophysiology of Decompression Sickness. The fundamental cause of decompression sickness is the evolution of bubbles within tissues and vessels. Tissues with an increased nitrogen content must release nitrogen to the blood which transports it to the lungs for elimination. When the ambient pressure is reduced too much or too rapidly, some tissues cannot diffuse their nitrogen into the blood fast enough and they become supersaturated, resulting in some nitrogen coming out of solution as bubbles. These bubbles can be interstitial, intralymphatic, or intravascular and have their effect either mechanically (as they obstruct blood flow or distort tissue) or indirectly (through endothelial damage, protein denaturation, and altered blood coagulation).

Although most cases of decompression sickness are seen after SCUBA diving, a reduction in ambient pressure from 1 atmosphere to less than one-half of an atmosphere (a rapid unpressurized ascent from sea level to an altitude greater than 18,000 feet above sea level) can also result in decompression sickness. The mechanisms are identical and the symptomatology varies only slightly.

The mechanical bubble effects are most prominent in spinal cord, brain, pulmonary, and, possibly, inner ear as well as peri-articular (pain only) decompression sickness. Analysis of spinal cord decompression sickness in dogs has revealed the following sequence:(*21,22,23*)

- Accentuated by slowed venous return, bubbles accumulate and coalesce in the epidural venous plexus.
- Aided by an increase in blood coagulability (see below), these bubbles

TABLE 1. Decompression Sickness (DCS) Classification and Comparison With Gas Embolism

Disorder	Symptom Complex	Pathology	Treatment
Type I - "Pain Only Bends" DCS	Pain confined to an extremity, usually peri-articular, aggravated by movement often relieved by direct pressure (i.e., with sphygmomanometer cuff)	Bubble evolution within non-distensible tissue (ligaments, tendons, bone)	Table V Recompression Observe for serious sx
Skin Pruritis	Intense itching, begins during decompression or shortly thereafter, primarily with air dives in chamber, direct pressure may relieve	Gas forced into sweat and sebaceous glands during dive evolves as bubbles on ascent (not true decompression sickness)	No Treatment
Skin Marbling	Itching, discoloration, cyanosis over affected area, blanches on pressure	Subcutaneous bubble evolution causing local venous stasis	Table V Recompression Observe for serious sx
Lymphatic Symptoms	Lymph node pain and swelling, tissue edema distal to affected nodes	Bubble accumulation within nodes, mechanical obstruction	Table V Recompression Observe for serious sx
Type II - All Other Pain DCS	Pain elsewhere other than extremities	May represent referred pain from visceral sites or spinal cord involvement	Table VI Recompression Observe for other sx
Central Nervous System	*ANY* hard or soft neurologic symptom; paralysis, sensory deficit, seizures, extreme fatigue, change in personality, headache, visual symptoms, etc.	Bubble evolution or accumulation anywhere in brain, spinal cord, or cranial nerves	Table VI Recompression
Labyrinthine	A CNS symptom also known as "staggers": any 8th nerve symptom; i.e., unsteadiness, vertigo, tinnitus, deafness, nystagmus	Bubbles obstructing labyrinth nutrient arteries, endolymph bubbles	Table VI Recompression
Pulmonary (Chokes)	Increasing substernal burning pain begins within minutes of surfacing, cough and progressive dyspnea follow; often associated with respiratory failure and shock	Accumulation of bubbles within pulmonary arterial tree	Table VI Recompression
Gas Embolism	Any CNS symptom on ascent or *within 10 minutes after surfacing*, can be rapidly progressive to circulatory collapse	Alveolar overpressurization with resultant free arterial air in cerebral and coronary circulation	Table VIA Recompression Life support measures as required

progressively obstruct epidural vertebral veins.

- Accumulation of bubbles in the pulmonary vasculature causes an increase in pulmonary artery pressure, increased mean central venous pressure, and increased central venous pressure fluctuations in response to varying intrathoracic pressure. When transmitted to the epidural veins, these pressure changes further slow blood flow and enhance obstruction of the epidural veins by bubbles.
- Venous cord infarct eventually occurs. Hemorrhagic white matter infarcts often occur at multiple levels with relative sparing of gray matter. (*24,25*)

The mechanical bubble effects that produce pulmonary decompression sickness are attributed to accumulation of gas bubbles in the pulmonary arterial tree. Doppler studies have demonstrated the presence of pulmonary bubbles in asymptomatic men and in decompressed dogs who, although asymptomatic, have increases in pulmonary artery and pulmonary wedge pressures, increased pulmonary vascular resistance, and decreased cardiac output.(*26*) With further bubble accumulation, tachypnea occurs early and is closely followed by increases in pulmonary artery systolic and diastolic, right ventricular systolic, and right ventricular end diastolic pressures, as well as decreases in cardiac output and arterial PO_2.*(27,28,29)* Decreased dynamic compliance and increased pulmonary vascular resistance have also been reported.*(30)* In addition, symptoms similar to those seen in pulmonary decompression sickness have been produced by inadvertent venous injection of air.*(31)* Although adult respiratory distress syndrome (ARDS) has been associated with pulmonary decompression sickness, it is distinctly uncommon.*(32)*

Type I (pain only) decompression sickness has also been attributed to the mechanical effect of gas bubble evolution within nondistensible tissue such as tendons and ligaments.*(33)*

Although most of the clinical manifestations of decompression sickness are attributed to the mechanical effects of bubbles as they obstruct blood flow and distort tissue, there are also many indirect effects caused by the blood-bubble surface activity interaction. Globular plasma protein denaturation has been reported,*(34,35)* which results in release of free phospholipids, cholesterol, triglycerides, and free fatty acids. Since coalescence of these lipids into globules has been observed, these globules may contribute to vascular obstruction.

Investigators have demonstrated an increase in the number of free circulating endothelial cells*(36,37)* and endothelial cells involved in platelet clumps*(36)* as well as histologic evidence of focal endothelial cell loss*(37)* in swine decompression sickness. The denuded areas may be sites for platelet adhesion and fibrin deposition, further contributing to vascular obstruction.

Studies of blood coagulation changes in decompression sickness have produced many interesting findings. Bubbles accelerate clotting in whole blood and cell-free plasma in vitro as a suspected result of Hageman factor activation.*(38)* A fall in circulating platelets, alterations in the PTT and factors V and VIII, and an increase in fibrin split products have been demonstrated after decompression in rats.*(39)* Platelet and fibrinogen consumption increase even in asymptomatic divers.*(40,41,42,43)* An intravascular bubble acts as a nidus for platelet adherence and aggregation, further obstructing blood flow.*(40)* Coating of red blood cells with denatured protein can cause red blood cell clumping, thereby increasing blood viscosity and promoting stasis.*(44)* Since venous blood flow is already slow, any further slowing could result in venous clotting with a consequent increase in capillary filtration pressure leading to interstitial edema and loss of plasma volume—a vicious cycle! *(44,45)*

Most cases of decompression sickness which are treated with recompression relatively early show rapid and complete improvement of all symptoms and recover without residua. This indicates that the indirect effects described above do not occur rapidly. These effects are important, however, in those patients who are left with permanent sequelae, either because treatment was delayed or the pathologic insult was too great. There is also a middle spectrum of patients who have slowly resolving symptoms; in these patients, the indirect effects had probably begun prior to treatment and their pathologic course requires time to reverse.

Decompression Sickness Treatment. Early recognition and treatment of decompression sickness is essential for resolution without sequelae. Even divers stricken with the most severe effects of decompression sickness can recover totally with prompt recompression, while delay can result in lifelong disability.

Experience has shown that aggressive initial treatment can significantly contribute to successful recovery. Patients with Type I decompression sickness will only require reassurance, oral fluids, and immediate transport to a recompression facility, but patients with Type II usually will require early, more vigorous supportive and therapeutic measures. The initial treatment guidelines for Type II differ only slightly from those for arterial gas embolism:

- Assure adequate airway, ventilation, and circulation. *NOTE:* Endotracheal tubes or foley catheter cuffs must be filled with water to avoid volume changes during recompression treatment.
- Administer 100 percent oxygen by the most efficient means (rebreathing mask if not intubated).
- Treat dysrhythmias or cardiac arrest with the ACLS protocol; dysrhythmias caused by bubble evolution into coronary arteries will tend to be refractory to treatment until the bubble is reabsorbed or reduced in size by recompression.
- Treat hypotension with pressors such as dopamine in doses of 1-10 ug/kg/min to increase coronary artery blood flow, cardiac output, and renal blood flow without increasing myocardial oxygen demand.
- Administer a bolus of dexamethasone (10-12 mg IV) followed by a maintenance dose of 4-6 mg every 6 hours.
- A foley catheter should be inserted if the patient is unconscious or there is urinary retention.
- Intravenous crystalloid with glucose should be started through a large bore catheter at a rate of 250-500 ml/hour; plasma expanders will also help prevent some of the effects of hemoconcentration and thrombus formation. Fluid input and output should be monitored.
- Valium may be administered in 5 mg IV boluses to a maximum dose of 30 mg for control of seizure activity as well as for sedation and to increase compliance with an endotracheal tube. Barbiturates are the drugs of second choice.
- Hypertonic solutions such as mannitol should *NOT* be routinely used because of the risk of a rebound increase in intracranial pressure. Their use should be reserved for cases of impending herniation.
- Transport the patient in the supine

position (see Part 1 for amplifying information). Ground transportation is preferred but, if air transportation is used, cabin altitude pressurization should be kept as close to sea level as possible or below 500 feet altitude above the site of injury for helicopters and unpressurized aircraft, consistent with safety of flight.(*46*)

Recompression is the mainstay of decompression sickness therapy and, although the details of recompression therapy will be handled by a specially trained treatment team at the recompression facility, the brief description which follows is presented as an overview. The U.S. Navy Diving Manual(*16*) is the authoritative guide for treatment of decompression sickness and details the use of a variety of recompression profiles. The vast majority of treatments are performed using either Treatment Table V or VI in dry chambers, most of which can accommodate a couple of patients as well as the mandatory attendant. These tables describe recompression chamber operations which bring the patient to an initial treatment depth of 60 FSW (2.8 ATA) where 100 percent oxygen is breathed (usually for 20-minute periods separated by 5-minute air breathing breaks to prevent pulmonary oxygen toxicity). After a variable time, a slow transition is made to 30 FSW where oxygen and air breathing is again alternated. After a variable time at 30 FSW, the patient is slowly brought to sea level pressure. The difference between Table V and Table VI is the length of time the patient remains at the 60 and 30 FSW depths; a Table V treatment is usually completed in less than 140 minutes whereas Table VI requires at least 270 minutes.

The three objectives in recompression therapy are to (1) reduce the size of the bubble, (2) promote bubble reabsorption, and (3) prevent further bubble evolution. Reduction in bubble size is important initially and is accomplished purely by the increase in ambient (chamber) pressure and results in relief of vascular obstruction and tissue distortion, thereby enhancing reperfusion and oxygenation. Intravascular gas is compressed into a spherical shape and reduced in size, promoting distal migration and reduction in the size of the ischemic area. (*47,48,49*) Bubble reabsorption is also augmented by the increase in ambient pressure as the partial pressure of the nitrogen within the bubble exceeds the partial pressure of nitrogen in the surrounding tissue, increasing the diffusion drive.(*50,51*) Breathing 100 percent oxygen washes out tissue nitrogen, enhancing diffusion by further widening the nitrogen partial pressure difference between the tissue and the bubble.

Based on the patient's response to recompression treatment, the tables are often modified by the chamber operators, usually lengthening them to provide added treatment time. Occasionally, treatment must be extended so long to prevent symptom recurrence that movement from 60 FSW to 30 FSW or from 30 FSW to the surface cannot be done without risking further bubble evolution. In this case, the dive may become a "saturation dive" which can be extended almost indefinitely at any depth, terminating with a very slow ascent accomplished over many hours or days. Multiple treatments are often required if symtpoms persist; in which case, therapeutic benefit is primarily derived from tissue hyperoxygenation rather than the hyperbaric effects.(*52,53,54*)

Adjuvant drugs are used during recompression treatment. These drugs should never substitute for recompression and are not necessarily useful in each case. Although many drugs have theoretically beneficial actions, their usefulness in the complicated pathologic process of decompression sickness should not be assumed. Oxygen has been unequivocally shown to be of significant benefit by enhancing tissue oxygenation, reducing cerebral edema, and washing nitrogen out of tissues to increase the bubble/tissue diffusion gradient.

Intravenous fluid therapy is a necessary adjunct in all cases of Type II decompression sickness because fluid loss, hemoconcentration, and increased blood viscosity promote vascular occlusion.(*44,45,55,56,57*) A crystalloid such as Ringer's lactate or normal saline with glucose can be used initially, the fluid for volume and glucose as a metabolic substrate. The fluid replacement in normotensive patients should be delivered at 250-500 ml/hour while monitoring blood pressure, hematocrit, and urinary output. Fluid replacement should not be so vigorous that pulmonary edema results from fluid overload. If hypotension arises, colloid replacement should be considered based on the clinical picture, hematocrit, and urinary output which should be at least 0.5 ml/kg/hour. Dextran has been successfully used as a volume expander, and it also has an anticoagulant effect. Both dextran 40 (40,000 molecular weight) and dextran 70 (70,000 molecular weight) have similar volume expanding properties; dextran 40 acts slightly more rapidly than dextran 70 but is more rapidly cleared by the kidneys.(*58,59*) Both dextran 40 and dextran 70 cause decreased platelet adhesiveness, decreased levels of platelet factor 3, decreased vascular sludging, and act to coat platelet, red blood cells, and vascular endothelium. (*58*) The use of dextran, however, is controversial(*57*) and, although low, the risk of side effects such as fluid overload, renal failure, and anaphylaxis are of concern.(*58,59*)

Since they are effective in trauma and brain tumor cases, steroids are recommended to decrease brain and spinal cord swelling.(*60,61*) Dexamethasone should initially be administered as an intravenous dose of 10-12 mg, followed by 4 mg either IV or IM every 4 hours. Unfortunately, the efficacy and exact dosage regimen has not been experimentally or clinically established for decompression sickness.

Shock associated with decompression sickness should be managed initially with fluids since the underlying pathology is intravascular fluid loss and loss of spinal cord control of vasomotor tone. Ephedrine in a 10-15 mg bolus may successfully temporize during transport to a chamber. Dopamine can be used in the 1-10 ug/kg/min dosage range to increase coronary artery blood flow, cardiac output, and renal blood flow without increasing myocardial oxygen demand. If shock persists despite dopamine therapy, epinephrine, neosynephrine, or levophed may also be used.

Although valium may cause respiratory depression, it is useful in controlling vertigo, nausea, and vomiting associated with labyrinthine decompression sickness. It will also help control seizures, decrease patient agitation, and increase compliance with intubation. Valium should be used with caution since it may delay treatment by masking a progression of labyrinthine symptoms.

Antiplatelet agents such as aspirin and dypridamole (and their analogs) may be effective in prevention of decompression sickness because of their actions on platelet kinetics but their efficacy in treatment has not been confirmed.(*41,62,63,64,65*) The use of heparin and other anticoagulants has been examined by many investigators (*39,56,62,66,67,68,69,70,71*) with inconclusive results. In cases of labyrinthine decompression sickness, anticoagulants (including dextran) are contraindicated since hemorrhage is thought to be important in its pathogenesis.(*70*)

After Treatment. Patients should be admitted to a hospital for overnight observation following any recompression treatment. *Any* recurrent or new symptom following treatment is automatically classified as Type II, a serious symptom, and the treatment is recompression on Table VI. Steroid therapy should be continued for 5-7 days with gradual tapering. The patient should not dive for at least 1 week after a Type I hit and at least 30 days following a Type II hit. Repeated Type I hits should not preclude reexposure, but a second Type II episode should warrant critical evaluation of the patient's fitness for further diving!

Use of an Alternate Inert Gas. To extend deep dive times, professional divers often use helium instead of nitrogen as the inert gas component of the breathing mixture. Helium is light, is easy to breathe, and has low solubility and high diffusivity, therefore making it easy to eliminate and supersaturation less likely to occur.(*72*) Decompression sickness can occur with helium-filled bubbles but less often than with nitrogen on the same dive profile. The use of helium also eliminates the problem of nitrogen narcosis (see below) during dives deeper than 100 FSW.

Another advantage of using a helium and oxygen mixture is that the relative concentration of oxygen can be varied, allowing lower oxygen concentrations to be used on deeper dives, diminishing the risk of pulmonary oxygen toxicity from prolonged exposure to oxygen at high partial pressures. The biggest disadvantages of using helium are that divers become easily chilled because helium enhances heat transfer and that communication is hampered by the Donald Duck-like helium voice.

Nitrogen Narcosis

Nitrogen narcosis or "rapture of the deep" is the progressive intoxicating or anesthetic effect of nitrogen which increases with depth. Each 50 FSW has informally been equated to having one martini: the "Martini Rule." With considerable individual variation, at 100 FSW, a diver can begin to feel lightheaded and euphoric, lose dexterity(*73*) and reasoning ability(*74*) and have increased reaction times.(*74*) A diver can become disabled by the disorienting and euphoric effects of nitrogen narcosis; he can lose his mouthpiece and be unable to find it, or even know which direction is up! Hypercapnia can accentuate the effects(*75*) and the only treatment is to ascend to a depth of relief. Divers who perform frequent deep dives breathing air can partially acclimatize to the narcotic effects of nitrogen. However, if deep dives are required, a better solution is to change the diluent to helium instead of nitrogen.

Shallow Water Blackout

A technique commonly used by free divers (snorklers who are not using compressed air or an accessory breathing device) to stay submerged for prolonged periods is to hyperventilate just prior to their dive. This eliminates a large amount of CO_2, decreasing arterial pCO_2—the primary respiratory driving force.(*76*) Decreasing the pCO_2 also results in cerebral vasoconstriction. As the diver descends, the increased ambient pressure results in a concomitant increase in arterial pO_2. Because the diver's respiratory drive has been suppressed, he tends to remain at depth until either his hypoxic drive or the eventual accumulation of CO_2 urges him to surface. The diver has remained conscious because the increased pO_2 at depth has maintained cerebral function but, as the ambient pressure rapidly decreases as he surfaces, the arterial pO_2 falls precipitously and, coupled with the cerebral vasoconstriction, can result in unconsciousness prior to reaching the surface. Shallow water blackout is most common among experienced divers who have become accustomed to holding their breath and tolerating excess arterial pCO_2 beyond normal limits. This potentially fatal problem can easily be prevented by not hyperventilating prior to a dive.

In Conclusion

Decompression sickness is a serious illness for which there is only one safe and efficacious treatment. Suspicion of decompression sickness should mandate recompression. Since the signs and symptoms are often subtle and confusing, any questions involving diagnosis or initial management should be promptly referred to the diving physician at a hyperbaric facility. In addition, 24-hour consultation is also available from the U.S. Navy Experimental Diving Unit in Panama City, FL, at (904) 234-4351, through Duke University's Diving Accident Network at (919) 684-8111, and from the Brooks Air Force Base hyperbaric treatment facility at (512) 536-3278.

References

1. Nims LF: Environmental factors affecting decompression sickness, in *Decompression Sickness.* New York, WB Saunders, 1951.

2. Elliott DH, Hallenbeck JM: The pathophysiology of decompression sickness, in Bennett PB, Elliott DH (eds): *The Physiology and Medicine of Diving and Compressed Air Work.* Baltimore, Williams & Wilkins, 1975.

3. Rivera JC: Decompression sickness among divers: An analysis of 935 cases. *Milit Med* 129(4):314-334, 1964.

4. Van der Aue OE, Kellar RJ, Brinton ES: The effect of exercise during decompression from increased barometric pressures on the incidence of decompression sickness in man. *US Navy Experimental Diving Unit Report,* Washington, DC, 1949, pp 8-49.

5. Bove AA: Effect of heat and cold stress on inert gas (^{133}Xenon) exchange in the rabbit. *Undersea Biomed Res* 5:149-158, 1978.

6. Tobias CA: Studies on skin temperatures and circulation in decompression sickness. *Am J Physiol* 149:626-633, 1947.

7. Cotes JE: *Influence of Age and Weight Upon the Incidence of Decompression Sickness in Personnel "Bends Tested" in the RAF Institute of Aviation Medicine,* DTIC AD-895-682. Defense Technical Information Center, Alexandria, VA, 1952.

8. Gray JS: Constitutional factors affecting susceptibility to decompression sickness, in *Decompression Sickness.* New York, WB Saunders, 1951.

9. Bassett BE: *Decompression Sickness in Female Subjects Exposed to Altitude During Physiological Training.* Preprints of Scientific Meeting, Aerospace Medical Association, Washington, DC, 1973.

10. Henry FM: Altitude pain: A study of individual differences in susceptibility to bends, chokes, and related symptoms. *Aviat Med* 17:28-55, 1946.

11. Hills BA: Biophysical basis of prevention and treatment, in *Decompression Sickness.* New York, J Wiley & Sons, 1977, vol 1.

12. Hills BA: *Fundamental Biophysical Aspects of Decompression Sickness,* DTIC AD-A069-998. Defense Technical Information Center, Alexandria, VA, 1979.

13. Balldin UI: Intracranial gas bubbles and decompression sickness while flying at 9,000m within 12-24 hours of diving. *Aviat Space Environ Med* 49:1314-1318, 1978.

14. Philp RB: Experimental analysis of the relationship between body fat and susceptibility to decompression sickness. *Aerosp Med* 35:351-356, 1964.

15. West VR: *A Review of the Influence of Physical Condition Parameters on a Typical Aerospace Stress Effect: Decompression Sickness,* DTIC AD-763-453. Defense Technical Information Center, Alexandria, VA, 1973.

16. *U.S. Navy Diving Manual,* NAVSEA 0994-LP-001-9010. Washington, DC, Government Printing Office, vol 1.

17. Kidd DJ, Elliott DH: Decompression disorders in divers, in Bennett PB, Elliott DH (eds): *The Physiology and Medicine of Diving and Compressed Air Work.* Baltimore, Williams & Wilkins, 1975, pp 470-492.

18. Kern JL: *The Etiology and Pathological Physiology of Decompression Sickness,* DTIC AD-260-755. Defense Technical Information Center, Alexandria, VA, 1960.

19. Strauss RH: Decompression sickness, in Strauss RH (ed): *Diving Medicine.* New York, Grune & Stratton, 1976, pp 63-82.

20. Dennison WL: *A Review of the Pathogenesis of Skin Bends,* DTIC AD-749-317. Defense Technical Information Center, Alexandria, VA, 1971.

21. Hallenbeck JM, Bove AA, Elliott DH: Mechanisms underlying spinal cord damage in decompression sickness. *Neurology* 25:308-316, 1975.

22. Hallenbeck JM: Cine photomicrography of dog spinal vessels during cord-damaging decompression sickness. *Neurology* 26:190-199, 1976.

23. Hills BA: Mechanical vs ischemic mechanisms for decompression sickness. *Aviat Space Environ Med* 50:363-367, 1979.

24. Haymaker W: Decompression sickness, in Labarsch O, Henke F, Rossle R (eds): *Hardbuch der Speziellen Pathologischen Anatomie und Histologica Nervensystem.* Berlin, Springer-Verlag, 1957, pp 1600-1672.

25. Kitano M, Hayashi K, Kawashima M: Three autopsy cases of acute decompression sickness. *J West Jpn Orthop Trauma* 26(3):110-115, 1977.

26. Neuman TR, Spragg R, Howard R, Moser K: Cardiopulmonary consequences of decompression stress. *Am Rev Respir Dis* 117 (4/2):162, 1978.

27. Bove AA, Hallenbeck JM, Elliott DH: Circulatory responses to venous air embolism and decompression sickness in dogs. *Undersea Biomed Res* 1:207-220, 1974.

28. Bergland E, Josephson S: Pulmonary air embolization in the dog: Hemodynamic changes on repeated embolizations. *Scand J Clin Lab Invest* 26:97-103, 1970.

29. McIver RG, Leverett SK: Studies in decompression sickness: Cardiorespiratory responses of anesthetized dogs to compression therapy following experimental decompression sickness. *Technical Documentary Report* SAM-TDR-63-94, School of Aerospace Medicine, Brook AFB, Texas, 1963.

30. Kahn MA, Alkalay I, Suetsusu, Stein M: Acute changes in lung mechanics following pulmonary emboli of various gases in dogs. *J Appl Physiol* 33:744-777, 1972.

31. Schmidt PJ, Kevy SV: Air embolism: A hazard during phlebotomy. *N Engl J Med* 258:424, 1958.

32. Ence TJ, Gong H: Adult respiratory distress syndrome after venous air embolism. *Am Rev Respir Dis* 119:1033-1038, 1979.

33. Inmann VT, Saunders JB: Referred pain from skeletal structures. *J Nerv Ment Dis* 99:660-667, 1944.

34. Lee WH, Krumbaar D, Fonkalsrud EE, Schjeido OA, Maloney JV: Denaturation of plasma proteins as a cause of morbidity and death after intracardiac operations. *Surgery* 50:29-39, 1961.

35. Lee WH, Huirston P: Structural effects on blood proteins at the gas-blood interface. *Fed Proc* 30:1615-1620, 1971.

36. Philp RB, Inwood MJ, Warren BA: Interactions between gas bubbles and components of the blood: Implications in decompression sickness. *Aerosp Med* 43:946-953, 1972.

37. Smith KH, Stogall PJ, Harker LA, Slichter SJ, Richmond VL, Hall MH, Haung TW: *Investigation of Hematologic and Pathologic Response to Decompression.* Office of Naval Research Technical Report N00014-71-C-0273, 1978.

38. Hallenbeck JM, Bove AA, Moquin RB, Elliott DH; Accelerated coagulation of whole blood and cell-free plasma by bubbling in vitro. *Aerosp Med* 44:712-714, 1973.

39. Inwood MJ: Experimental evidence in support of the hypothesis that intravascular bubbles activate the hemostatic process, in Ackles K (ed): *Symposium on Blood-Bubble Interactions in Decompression Sickness,* DCIEM Report 73-CP-960, 1973.

40. Philp RB, Ackles KN, Inwood MJ, Livingston SD, Achimastos A, Binns-Smith M, Radomski MW: Changes in the hemostatic system and in blood and urine chemistry of human subjects following decompression from a hyperbaric environment. *Aerosp Med* 43:498-505, 1972.

41. Philp RB, Inwood MJ, Ackles KN, Radomski MW: Effects of decompression on platelets and hemostasis in men and the influence of antiplatelet drugs (RA233 and VK744). *Aerosp Med* 45:231-240, 1974.

42. Philp RB, Freeman D, Francey I, Ackles KN: Changes in platelet function and other blood parameters following a shallow open-sea saturation dive. *Aerosp Med* 45:72-76, 1974.

43. Philp RB: A review of blood changes associated with compression-decompression: Relationship to decompression sickness. *Undersea Biomed Res* 1:117-150, 1974.

44. Hallenbeck JN: Some aspects of the basic pathophysiology of decompression sickness, in *Weekly Update: Hyperbaric and Undersea Medicine.* Princeton, Biomedia, vol 1, no 10, pp 1-8.

45. Bove AA, Hallenbeck JM, Elliott DH: Changes in blood and plasma volumes in dogs during decompression sickness. *Aerosp Med* 45:49-55, 1974.

46. Reddick EJ: Movement by helicopter of patients with decompression sickness. *Aviat Space Environ Med* 49:1229-1230, 1978.

47. Epstein PS, Plesset MS: On the stability of gas bubbles in liquid gas solutions. *J Chem Physic* 18:1505-1509, 1950.

48. Harvey EN, Barnes DK, McElroy WD, Whitley AH, Pease DC, Cooper KW: Bubble formation in animals: I. Physical factors. *J Cell Comp Physiol* 24:1-22, 1944.

49. Van Liew DH: Factors in the resolution of tissue gas bubbles, in Lambertsen CJ (ed): *Underwater Physiology, Proceedings of the Third Symposium on Underwater Physiology.* Baltimore, Williams & Wilkins, 1967, pp 171-204.

50. Hlastala MP, Van Liew DH: Absorption of in vivo inert gas bubbles. *Respir Physiol* 24:147-158, 1975.

51. Van Liew DH, Hlastala MP: Influence of bubble size and blood perfusion on absorption of gas bubbles in tissue. *Respir Physiol* 7:111-121, 1969.

52. Workman RD: Treatment of bends with oxygen at high pressure. *Aerosp Med* 39:1076-1083, 1968.

53. Yarborough OD, Behnke AR: The treatment of compressed air illness utilizing oxygen. *J Ind Hyg Toxicol* 21:213, 1939.

54. Kindwall EP: Decompression sickness, in Davis JC, Hunt TK (eds): *Hyperbaric Oxygen Therapy.* Undersea Medical Society, Bethesda, MD, 1977.

55. Cockett ATK, Nakamura RM: A new concept in the treatment of decompression sickness (dysbarism). *Lancet* 1:1101, 1964.

56. Kindwall EP, Margolis I: Management of severe decompression sickness with treatment ancillary to recompression: Case report. *Aviat Space Environ Med* 46:1065-1068, 1975.

57. Wells CH, Hilton JG, Hall CB: Microcirculatory effects of intravenous fluid resuscitation in dysbarism. *Undersea Biomed Res Supp* 5:18, 1978.

58. Data JL, Nies AS: Dextran 40. *Ann Intern Med* 81:500-504, 1974.

59. Childs C, Ah-See AK, Arfors KE: Dextran administration in the treatment of diving accidents. *Medecin Aeronautique et Spatiale, Medecine Subaquatique et Hyperbare* 17:83, 1978.

60. Faupel G, Reulen HJ, Muller D, Schurnamm K: Double-blind study on the effects of steroids on severe closed-head injury, in Pappius H, Reulen H (eds): *Second International Workshop on Cerebral Edema, Montreal 1976.* Berlin, Springer-Verlag, 1977.

61. Fishman RA: Brain edema. *N Engl J Med* 293:706-711, 1975.

62. Philp RB: The ameliorative effects of heparin and depolymerized hyaluronate on decompression sickness in rats. *Can J Physiol Pharmacol* 42:819-829, 1964.

63. Philp RB, Freeman D, Francey I, Bishop B: Hematology and blood chemistry in saturation diving: I. Antiplatelet drugs, aspirin, and VK744. *Undersea Biomed Res* 2:233-249, 1975.

64. Philp RB, Bennett PB, Anderson JC, Fidds GN, McIntyre BA, Francey I, Briner W: Effects of aspirin and dypridamole on platelet function, hematology, and blood chemistry of saturation divers. *Undersea Biomed Res* 6:127-146, 1979.

65. Clark ML, Philp RB, Gowdy CW: Seratonin and other vasoactive agents in experimental decompression sickness. *Can J Physiol Pharmacol* 47:1033-1035, 1969.

66. Cockett ATK, Pauley SM, Roberts AP: Advances in treatment of decompression sickness: An evaluation of heparin. *Third International Conference on Hyperbaric and Underwater Physiology,* Marseille, 1970.

67. Reeves E, Workman RD: Use of heparin for the therapeutic/prophylactic treatment of decompression sickness. *Aerosp Med* 42:20-23, 1971.

68. McCormick JG, Holland WB, Brauer RQ, Helleman IL: Sudden hearing loss due to diving and its prevention with heparin. *Otolaryngol Clin North Am* 8:417-430, 1975.

69. Farmer JC, Thomas WG, Youngblood DG, Bennett PB: Inner ear decompression sickness. *Laryngoscope* 86:1315-1327, 1976.

70. Barthelemy L: Blood coagulation and chemistry during experimental dives and the treatment of diving accidents with heparin. *Second Symposium on Underwater Physiology,* National Academy of Sciences, National Research Council Report Number 1181, 1963.

71. Saumarez RC, Bolt JB, Gregory RJ: Neurological decompression sickness treated without recompression. *Br Med J* 1:151-152, 1973.

72. Shilling CW, Werts MF, Schandelmeier NR (eds): *The Underwater Handbook: A Guide to Physiology and Performance for the Engineer.* New York, Plenum Press, 1976, pp 189-205.

73. Kiessling RJ: Performance impairment as a function of nitrogen narcosis. *US Navy Experimental Diving Unit Report* 3-60, 1960.

74. Shilling CW, Willgrube WW: A quantitative study of mental and neuro-muscular reactions as influenced by increased air pressure. *US Nav Med Bull* 35:373-380, 1937.

75. Hesser CM, Adolfson J, Fagraeus L: Role of CO_2 in compressed air narcosis. *Aerosp Med* 42(2):163-168, 1971.

76. West JB: *Respiratory Physiology—The Essentials.* Baltimore, Williams & Wilkins, 1974, pp 116-128. □

Internship Selections

Each year NAVMEDCOM receives about 500 internship applications for a little more than half that number of available positions. This provides particularly keen competition in the selection process. In arriving at decisions the committee has available only what your references have submitted in writing. This is a challenge that all of you need to accept and do something about. Here are some tips:

- When your internship application packet is received (about 1 May of the year preceding your graduation year), read it carefully and note the schedule of dates. Comply.
- You can assure that your personal submission is on time but there are others who must also meet deadlines on your behalf. Be sure that they do so and, if you can, assure that their evaluations are thorough and describe you as an individual. Believe it or not we have received letters from one source on several students that were form letters with only the names changed. To say the least, these were of dubious value to the selection committee.
- When you complete a Navy clerkship, speak to your graduate training office secretary about getting a copy of your evaluation forwarded to the Naval Health Sciences Education and Training Command (Code 14), Bethesda, for inclusion in your professional record. If you can, visit at least one hospital for an interview. These clerkship and interview reports are available to the selection committee.
- Because of your academic accomplishments, all of you will be acceptable candidates for training and most of you will probably be very desirable candidates for any specialty program of your preference. Rightfully, an interviewing officer at a given naval hospital may tell you this. Please remember that such statements of desirability are not tantamount to selection.

Caution: Fraud and substance abuse are not consistent with Navy policy and, at times, constitute criminal acts. We have recently experienced one case of drug abuse and one case of a documented fraudulent claim among our student community. The consequences have been very serious.

Deferments

The Navy's Active Duty Delay for Specialists (NADDS) program was established as an integral part of the Navy's overall training plan. Participants are granted deferments from serving active duty obligations while undergoing specialty training in the civilian sector. They serve without pay or allowances from the Navy and are free to accept the stipends and benefits paid to other trainees in their programs. Personnel eligible to be considered for deferments are:

- Graduating medical and osteopathic students *who are not selected for Navy internships.*
- Program participants who are deferred for civilian internship and *who are not selected for continued training Navy programs.*
- Interns (USNR only) *who are not selected for continued training in Navy programs.*
- All other Reserve medical officers on active duty *who are not selected for Navy training.*

There is no additional obligated service required for participation in the NADDS program. However, officers serving on active duty who are released for civilian training in a deferred status must sign an agreement that when recalled to active duty they will serve for a minimum period of 2 years. This is not considered to be an obligation but is a minimum term of service. Officers who are recalled and have at least 2 years remaining from a previous obligation are required to serve only that obligation.

While serving in the NADDS program officers remain eligible for promotion and many are considered by inactive Naval Reserve selection boards. Unfortunately, the records of most of this group usually contain very little information on which the board can base a determination as to the officers' fitness for promotion. Laws and regulations are very clear and restrictive as to what can be entered into an official record and by whom. In order to obviate the nonselection of the participant, based solely on a lack of performance reports in his/her record, the Naval Medical Command will submit not-observed performance (fitness) reports on all NADDS officers. Those reports will identify the officer, explain what he is doing and why. Selection boards may not select NADDS officers for promotion, but at least the board members will have an understanding of the officers status.

Each year all officers are required to reconfirm that they are training in an approved program in the specialty for which selected for deferment. Maintaining communication with this group is not difficult because we know where they are. However, medical students who go directly into the NADDS program and their first year of training is an internship and not a part of the specialty program are, at times, hard to find. It is very important that all obligated medical officers keep Code 544, Naval Medical Command, Washington, DC 20372-5121 advised at all times of their mailing address and the type, place, and inclusive dates of their training programs.

General

Commissioned naval officers can administer oaths and witness official documents in the conduct of official business. As a scholarship student or a member of the NADDS program, you are a commissioned officer. During your term in the Navy, it will be necessary that you sign documents (internship agreements, deferment agreements, etc.) that require a witness to your signature. This can be done by any commissioned officer, including a fellow scholarship student, or by a notary public. □

☆ U.S. GOVERNMENT PRINTING OFFICE: 1985 - 461-650 - 128/20300

DEPARTMENT OF THE NAVY
Naval Publications and Forms Center
ATTN: Code 306
5801 Tabor Avenue
Philadelphia, PA 19120

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE, $300

Second-Class Mail
Postage and Fees Paid
USN
USPS 316-070

MSC Anniversary Ball

The Navy Medical Service Corps will celebrate their 38th anniversary 3 Aug 1985 at the Bethesda Officers' Club, located on the compound of the Naval Medical Command, National Capital Region, Bethesda, MD. All Medical Department officers, active, Reserve, and retired, are invited.

This year's theme, "38 Years of Medical Service Corps Magic," highlights both the Corps' length of service to the Nation, and their worldwide structure and commitments to the Fleet.

Reservations are required. Tickets are $15.00 per person for WO/ENS/LTJG and guests, $20.00 per person for LT/LCDR and guests, $25.00 per person for CDR/above and guests, and $25.00 per person for civilians. For information, call LCDR Sheila Weinberg at (Autovon or Commercial) 295-0342.